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PULMONARY TUBERCULOSIS WITH ACUTE ONSET

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That pulmonary tuberculosis frequently has an acute onset, is a fairly new conception of this disease. Our early teaching was that phthisis has a very insidious onset, begins in the apex of the lung and spreads downward as it progresses; that adults are relatively immune from exogenous infection; that, if they develop tuberculosis, it is a reactivation of a previous childhood infection, or a metastasis from a reactivated primary lesion. In other words, it is endogenous rather than exogenous. With further research and the added knowledge that x-ray brings us, many of our pre-existing conceptions have been justifiably questioned.

Assman[1] in 1924 called attention to the onset of progressive disease as being infraclavicular, at the same time stating that true apical disease was generally benign and seldom was progressive. Redeker[2] followed with similar observations, and he is generally conceded to be the foremost exponent of the subclavicular onset of progressive tuberculosis. Authors throughout the world became interested in the subject, and there followed numerous articles calling attention to the fact that infraclavicular early infiltration had a more acute onset than had been our previous teaching of pulmonary tuberculosis.

Douglas, Pinner and Walepor[3] sharply differentiated between acute subapical and insidious apical involvement. However, it was not clear in their presentation what was their criterion for acute onset, as to time or acuity of symptoms. It was clear, however, that from their observation the commonest onset of pulmonary phthisis was more abrupt than our usual idea of it. They found the incidence of acute subapical onset to be 52 per cent. Also, attention was called to the fact that, in subapical involvement, rapidly progressive or retrogressive changes occur. Numerous publications since then have concurred in the opinion that subapical involvement is very labile. It gets better or worse soon after its onset.

Pulmonary tuberculosis with a very acute onset resembling influenza or pneumonia, is not infrequent. It occurs often enough that the possibility of its presence must be considered in diagnosis. Its importance is accentuated by the fact that early diagnosis can usually prevent progression to cavitation later. Relatively few acute cases come under observation early, because they are diagnosed only after the secondary exacerbation occurs, which is usually months after the onset.

In order to ascertain approximately just how frequent is the onset with symptoms like influenza or pneumonia, I reviewed the history files of patients seen since 1925. Histories previous to this date were rejected to avoid the influenza epidemic of 1918-1919, and subsequent years. Only cases of pulmonary tuberculosis with frank parenchymal involvement were used. Cases having an onset with hemorrhage, pleurisy with effusion, or a cold, were classified as insidious. Cases with onset with acute fever, pneumonia, or influenza, were considered acute, if the symptoms and ultimate diagnosis of tuberculosis were continuous with the acute onset. Histories whose type of onset was not clear, were rejected. In this survey 300 histories were reviewed. Acute onset, according to the above criteria, occurred in 33.9 per cent, slightly over one-third of these 300 cass.

Now, there is always a personal equation and considerable error in a review of this type. Also, a certain number of cases probably really were influenza or pneumonia which lighted up a dormant tuberculous focus. But, even considering this, there is a fairly high percentage with acute onset.

Similarity of tuberculosis with an acute onset to the so-called influenza, is obvious. They have in common, fever, aching, malaise, with no very definite localization of the diseased process, except perhaps a little cough. A patient's conception of influenza is fever and general aching over the body. One wonders how many cases of tuberculosis with acute onset are diagnosed influenza.

A certain percentage of patients with tuberculosis, date the onset to an attack of "flu," as they call it. Considerable doubt was aroused, as to whether influenza activates a latent tuberculous process, by our experience with the influenza epidemic of 1918 and 1919. Contrary to expectations, the mortality from tuberculosis steadily diminished, in spite of the epidemic. It was entirely unaffected by it. Fishberg[4] called attention to this fact. Quoting him: "Epidemic influenza has no etiological relationship to tuberculosis and is not to be considered as a reactivator of dormant tuberculous lesions. Vital statistics and reports from sanatoria show that, during the past and current years, the mortality from tuberculosis has not increased; nor has the number of patients with tuberculosis seeking admission to sanatoria increased." This statement brought forth a storm of protest from writers over the country, but its truth has been verified by subsequent vital statistics. These facts make one dubious as to whether the onset with influenza, in pulmonary tuberculosis, is really influenza at all.

The following case is a rather typical example of an acute onset. It is particularly instructive for it occurred in a physician specializing in pulmonary tuberculosis, who carefully observed symptoms.

CASE I. Dr. F. B., age, 40. Previously good health, very active in practice. Felt achy and feverish after hard day's work on Jan. 22, 1932. Next day, fever 102°, without any symptoms except fever. Physical examination, after three days, revealed nothing. Fever 104° next few days, then receded to 102.5°. At end of seventh day, still no physical findings, except fever, and no symptoms except those of a febrile condition. Went to Presbyterian Hospital, Chicago. X-ray on tenth day, revealed consolidation of right upper lung area. A few fine rales were heard over the same area, at this time. Still no cough or sputum. Fever 99.6° daily. Diagnosis was made of acute pulmonary tuberculosis. Fever gradually subsided with rest. Never had cough or sputum. Gradual uneventful recovery.

Here is an abrupt onset, clinically resembling pneumonia, the diagnosis of which could not be accomplished without the x-ray, for the history and physical findings gave little suggestion of the lungs being the source of the infection, until rales were heard, ten days after onset. The progress of this case was one of rather prompt resolution, without cavitation and with little fibrosis. The origin of the metastasis was not obvious; probably it was from the rupture of a caseous hilum gland into a bronchus.

CASE II. Mrs. R. P., age 26. Good health until Thanksgiving, 1931, when exposed at football game; following this, began to cough some. Kept at her usual activities. About Jan. 1, 1932, became very ill, fever 102°. In a few days, the patient was delirious, with high fever. Fever continued. X-ray diagnosis, Jan. 27, 1932, was "probable lung abcess with dense mottled infiltration at base of right lung." Fever less than 101° daily, until March, and since then 99.6° daily. Up and about since March, decreasing cough and raising. X-ray in Tucson, Sept. 10, 1932, reveal marked reduction of area of infiltration in right base; but in the right upper lung area, there is fine closely applied mottling. Tubercle bacilli were found in sputum soon after this date. Tuberculous infection in child of 3 years old.

Here is probably a bronchogenic metastasis resulting in a large basal tuberculous pneumonia. The diagnosis was not certain until tubercle bacilli were found in the sputum eight months later and infection in the right upper lung area could be easily seen. The ultimate outcome of this case is still uncertain. However, it is noteworthy that cavitation was neither obvious nor early. There was a marked tendency to resolution in spite of little or no treatment.

Case No. 3 is an example of an acute onset of an extension from a known apparently arrested focus.

CASE III. J. G., age 42. First diagnosed active tuberculosis in the summer of 1930, although he had been having pulmonary symptoms off and on for five years previously. Came under observation December, 1930, with fibrosis and cavity in left upper lung. Cavity closed by February, 1932. On graduated exercise, in the spring and summer of 1932, the patient was quite well. No symptoms. Suddenly, July 10, 1932, he had a fever of 101°, next day 103°. Acute fever about ten days, gradually subsiding. No unusual findings in lungs, no dullness or rales. X-ray, taken July 27, 1932, revealed large extension in the left lower lung. With four months' rest, this has largely subsided, leaving only the findings of thickened pleura in this area.

Here was an abrupt onset in a few days only, closely resembling the onset of pneumonia, without symptoms referable to the lungs and without any physical findings of new involvement. No abnormal pulmonary findings were present at any time during the course of the new involvement. This case differs little from the two preceding ones, except in having previously an obvious infection. Contrary to Ranke's classification, this case, although tertiary, was apparently highly allergic.

The clinical characteristics of pulmonary tuberculosis with acute onset are:

1. Onset closely resembles pneumonia and influenza.

2. Rapid retrogressive changes occur with strict rest.

3. Physical signs are absent or relatively few.

4. Few localizing symptoms, as cough or pain in the chest, are present.

There is a certain parallelism, in these cases of acute onset, to the work of Soper, Sampson, and Hopkins[8] on aspiration tuberculosis in rabbits. Part of their conclusions are:

1. "An early pneumonic reaction to aspiration of virulent tubercle bacilli may occur in the lungs of both sensitized and normal rabbits. It is more immediate, more intense, and more constant, in the sensitized rabbits."

2. "Of the different factors which determine the degrees of early reaction to tubercle bacilli in the sensitized lungs, the quantity of bacilli introduced is a very important one."

3. "The initial pneumonia reaction may undergo almost complete resolution in both sensitized and normal rabbits. Resolution is more rapid, more complete, and more constant, in the sensitized animals."

The marked similarity of adult human lungs to rabbit lungs in their response to tuberculous infection, has been pointed out by Krause[6] previously. This suggests that sensitization favors more rapid resolution in aspiration infections in human adult lungs.

A distinct impression is given that patients with an acute onset resembling pneumonia, heal more rapidly than others with a more gradual onset. This again suggests that the former type has a higher relative immunity. That there is a marked degree of allergy or hypersensitiveness in the cases presented, seems clear, considering the marked febrile response and the presence of an exudative type of involvement. Quoting Krause[7], in his work on experimental tuberculosis:

"Again we note that symptoms of acute illness occur only in reinfected animals and never in controls. This suggests, what is probably true, that acute tuberculosis never develops as a first infection, that it is acute because it is more of the exudative type, and that its exudative type and the acute symptoms are simply expressions of resistance of over action, of immunity, of hypersensitiveness, call it what you will."

The explanation of the acute onset can be either: (1) the presence of a high degree of tissue hypersensitiveness alone, or (2.) A rather massive bronchogenic infection plus a high degree of allergy. We know that in both adults and children there is a rather wide variation in the response to the intracutaneous injection of tuberculin; hence, different degrees of the allergic state. This degree of allergy is mainly determined by the amount and activity of the existing infection.

The relationship of allergy and relative immunity is not entirely clear. They have many features in common, however. There is little convincing evidence that they are two separate phenomena in tuberculosis.

Krause[6] in a paper on this subject states:

"As regards immunity: (1.) There is no specific immunity without pre-existing tubercle. (2) Immunity first appears with the palpable development of the focus. (3.) It diminishes with the healing of the focus. (4.) It disappears with the enucleation of the focus. (5.) The degree of immunity varies directly with the virulence of the immunizing microorganisms or with the amount of tubercle and the activity of lesions in the host.

"As regards allergy: (1.) It is never observed unless tubercle is present. (2.) It appears coincidently with the establishment of lesions. (3.) It diminishes as lesions heal. (4.) It varies directly with the intensity of the disease, which in turn is dependent, not wholly, but in large measure, on the virulence of the bacilli concerned."

As this pertains to acute onset, it is probable that there has been recently, or there coexists, an active focus which makes the host highly allergic, and that this focus of caseation ruptures into a bronchus, resulting in a sudden planting of tubercle bacilli and tubercle protein in highly sensitized healthy lung.

That individuals vary as to their sensitiveness to tuberculin, is an accepted fact. So, it is not unreasonable to conceive of different responses to reinfections or new infections. Perhaps the whole phenomenon of acute onset can be explained on the basis of the amount of infected material planted, namely, a pathological happenstance, sending a large bulk of infection into healthy allergic lung tissue, not dependent on an unusually high degree of hypersensitiveness at all.

Healing of a tuberculous process both in the lungs and peritoneum, without leaving any x-ray or gross pathology, has been a common observation. Un-

questionably, cases of less extent than here shown, entirely clear, leaving no tangible evidence of their former presence. The common finding of a healed area of tuberculosis in the lungs, without any history of pulmonary disease, probably could be explained on the basis of an acute onset of tuberculosis diagnosed as influenza, which subsequently healed.

I have no doubt that tuberculosis with acute onset will have a less favorable outcome than the cases here presented, if it is incorrectly diagnosed, and the patient is allowed to pursue his usual mode of living soon after the fever subsides.

Concerning early diagnosis of pulmonary tuberculosis, if the diagnosis is made on symptoms, physical signs, and tubercle bacilli in the sputum, these cases usually have already progressed to excavation. The earliest diagnosis must be made with the x-ray film.

The more frequent use of roentgenograms of the lungs in obscure fevers, and in cases with indefinite symptoms, will enable us to make the earliest possible diagnosis, while the prognosis is still favorable, and avoid the secondary exacerbation with its early cavitation.

SUMMARY AND CONCLUSIONS

1. Three cases of pulmonary tuberculosis with acute onset like influenza, are presented.

2. The incidence of acute onset from a review of 300 histories, was 33.9 per cent.

3. It is suggested that the great majority of acute cases are incorrectly diagnosed influenza, until secondary exacerbation and excavation have occurred.

4. X-ray offers the earliest means if making a correct diagnosis.

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WHAT CAN BE DONE FOR THE FAR ADVANCED CASE OF PULMONARY TUBERCULOSIS

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I am unable to bring you anything new in the field of phthisiotherapy unless it be a new enthusiasm.

I am not a surgeon, but it is to the thoracic surgeon that I am indebted for this new enthusiasm for what can be done for the far-advanced case of pulmonary tuberculosis. In no other field of medicine have the clinician and surgeon been brought so close together and learned to cooperate with, and depend upon, each other. The internist must select the patient, make the necessary study of the case, correlate the physical findings and x-ray and all other necessary studies, in preparation for the work of the surgeon. Internist and surgeon have thus learned from experience in this field of collapse therapy that the greatest success depends upon cooperation with, and helpfulness to, each other. They have thus been able to lift a great group of individuals from a condition of despair to that of hope, prolonged life, and healthfulness.

As clinical director of one of the government's tuberculosis, general medical and surgical hospitals, it is part of my function to correlate this kind of work. I have been in the government work since 1918, and most of that time has been devoted to the study and treatment of tuberculosis.

Immediately after the war, I saw a great group of young adult males suffering with active pulmonary tuberculosis, either with new lesions or reactivations of old, healed, or quiescent lesions. I saw this group of young men treated in various institutions by the usual hygienic measures, including the triad of rest, fresh air, and good food. At no time have I seen such remarkable results as were obtained at that time.

A good many of these men, when placed on the usual rest regime, immediately lost their toxic symp-

toms, made remarkable gains in weight, tuberculous lesions improved rapidly, and many went on to arrest of their lesions. I became quite enthusiastic over the great number of rapid improvements and arrests; however, as time went on and I continued this tuberculosis work and followed this group of veterans from year to year, I began to realize that fewer and fewer of them went on to arrest or cure. Now, in 1932, I find our hospital filled with practically only cases of advanced, adult, pulmonary tuberculosis, most of whom are infirmary cases, and those who are ambulant have extensive lesions. Most of the cases are far advanced, with cavitation—men who have been treated in various government and civilian institutions and at home for years; men who have met failure with treatment, without treatment, with inadequate treatment, or in spite of treatment.

As I have observed these cases progress into cavitation and chronic invalidism from year to year, I must confess that some of my former optimism was replaced by discouragement. While in the early years following the war we strove for industrial rehabilitation, now we more and more hoped for prolongation of life and some measure of social rehabilitation. Only comparatively recently have I really become alive to the therapeutic possibilities of the various methods of collapse therapy, even in these far advanced cases; cases which we hitherto believed hopeless. True, we had always done a certain number of artificial pneumothoraces in all of our hospitals, enthusiasm for this form of therapy varying widely among the staffs of the different hospitals. It has been only recently, with the advent of phrenicectomy, thoracoplasty, and the more general and liberal use of pneumothorax, that I again begin to feel satisfied with my work with these far-advanced, tragic cases.

The majority of our cases have cavitation, and it has been our experience that, as long as these cavities persist, the patient is doomed to a life of invalidism, with positive periodical sputum, exacerbations, extensions or hemorrhages, and with a life expectancy of only a few years. While many of these cavities heal spontaneously with the usual hygienic treatment, there remains the great group which has met failure with this regime and for whom some form of collapse therapy is demanded. Collapse therapy in no way replaces our usual rest and hygienic routine for the tuberculous, but is an adjunct thereto. The splinting and immobilization of the lung, by either partial or complete collapse therapy, is truly rest therapy analagous to the splinting of a joint tuberculosis. The compression of a diseased lung or of a large cavity produces marvelous results which prolonged rest alone can never attain. The management of the far-advanced case has entirely changed and now we feel gratified that we can do more for this type of case. No longer do we have a feeling of hopelessness and despair.

In some places I find the old idea of ultra-conservatism still existing in regard to collapse therapy, but I believe we should not continue to follow precedent unless it can stand the test of comparison with methods necessary to meet changing conditions.

My own experience in regard to pneumothorax therapy is amusing to me when I look back a few years, and serves to illustrate the common error of following precedent. I can remember, not so long ago, that in selecting cases for pneumothorax I found only few who met my demands for unilateral pneumothorax. I demanded a strictly unilateral involvement. The contralateral lung must be absolutely clear, especially from any active involvement. The presence of any rales in contralateral lung surely precluded further consideration.

My opinion, like the opinion of other physicians and of the laity as well, was that the contralateral lung, if diseased, could not stand the extra load which would be thrown upon it by collapse of its sick partner. I had heard that expression so many times from doctors and from patients that I adopted it as gospel truth and, by so doing, denied many men a chance for future health. Sooner or later, many of these advanced bilateral cases had severe pulmonary hemorrhages, perhaps uncontrollable hemorrhages. All the usual remedies and procedure having failed, artificial pneumothorax was given on the cavity side as a last resort and as a life-saving measure.

After repeated experience of this kind, we finally began to awake to marvelous things. In some of these patients we not only checked the hemorrhage, but something more had happened. Later, the temperature had dropped, the patient rested more comfortably, his general well-being was improved and, when checked by x-ray, a still more marvelous thing had happened. The contralateral lung, which had considerable involvement, was much improved, there was actual absorption and clearing of lesion, so much so that pneumothorax was continued and, to our surprise, we began to lose faith in our old tradition; namely, that the sick partner would not be able to carry on the work of its collapsed fellow. Now, we are convinced that we are not throwing an extra load on the contralateral lung, but, in reality, we are removing a load from it. We now know that, when we remove that big load of absorption and toxicity,

by collapse of the worst cavity lung, then the contralateral lung, although sick itself, will often entirely cure itself.

Now, we no longer demand one good lung in choosing cases for unilateral pneumothorax, the clearing of the disease in the contralateral lung being oftentimes so remarkable that we are forced to wonder if there are not some other factors involved outside of the mere compression of tuberculous lesion and cavities and reduction of toxemia. It has been suggested that the compression of the pulmonary lesion causes an auto-tuberculinization and it is because of this increase in tuberculin sensitization that we sometimes see such remarkable clearing of parenchymal lesion, and even cavity closure, in the contralateral lung.

We used to believe, and many are still of the opinion, that we had to entirely collapse a cavity to obtain closure; however, we now know that only partial collapse, especially in case of thin-walled cavities and cavities centrally located, is all that is necessary. To be convinced of this, we need only to look at the many cases of complete cavity closure which has resulted in partial collapse therapy with phrenic nerve exeresis.

We used to make dogmatic decision as to whether or not cases could obtain collapse, by examining x-ray plates and by physical examination. We were proud of our expert guessing ability. Now, I find that many cases which hitherto I was confident could not be collapsed, on account of pleural adhesions, can be collapsed when tried. I now make a practice of denying no patient at least an attempt at pneumothorax because of my preconceived ideas of adhesion, pleural thickening, and so forth. I have seen successes too many times in such cases. The patient is entitled to repeated trials.

It was formerly our practice, and is still the practice of many, the moment a new lesion developed in the contralateral lung, to become alarmed and discontinue collapse therapy; likewise, if we did not obtain complete cavity closure and complete results as we expected, we would discontinue pneumothorax. From experience, we have learned that the patient has more chance for recovery by continuance of the pneumothorax, and that when pneumothorax is discontinued the lesion goes on to further progression; also, that lives are prolonged and that life is made more comfortable by continuance of a partially successful collapse. This class, even if it refuses further surgical collapse therapy, such as thoracoplasty, should be continued indefinitely.

The increasing problem of far-advanced, bilateral tuberculosis has forced me to adopt, in certain cases, a more heroic form of pneumothorax—that is, bilateral, artificial pneumothorax. It is my opinion that this form of therapy is not being used to the extent it should be. Many workers are allowing fancied but dreaded complications to cause them to withhold this very useful therapy. We believe that the prolongation of life, control of hemorrhage, and remarkable gains which these patients make far outweigh all the hazards of bilateral pneumothorax. Several case reports with x-rays, which I shall show you, will, I am sure, convince the most skeptical.

There are several points, however, in connection with bilateral pneumothorax, which I would emphasize. It is necessary for the operator to realize that only partial or selective collapse can be induced or is necessary. He must know that a partial collapse with a constant negative pressure will cause closure of cavities and, by immobilization of lungs, will heal parenchymal lesions. He must maintain a fairly equal pressure over both lungs, especially in cases with movable mediastinum. He must not attempt this form of therapy unless he is prepared for minute study and close observation of his patient. He must be prepared for frequent x-ray study of his case; fluoroscopy alone is not enough. This form of therapy should preferably be carried out in a hospital with facilities for proper observation and study. I have cases at our hospital at this time under this form of therapy, all far-advanced, bilateral cases with bilateral cavitation—you might say terminal cases; cases which would ordinarily be considered hopeless. They gain weight, lose their cough and expectoration. The result is little short of miraculous. We are becoming so encouraged by our recent experience in this field that we are routinely canvassing our wards for cases suitable for this form of therapy.

We have become quite enthusiastic over the benefits to be derived in these far-advanced cases by the more general use of the simple operation of phrenic nerve evulsion. Here, as in the use of pneumothorax collapse therapy, our views on the selection of cases and the benefits to be derived, have changed after several years of experience. At first, we chose only unilateral cases, with basal parenchymal lesion and cavitation in lower lobes. We accepted the traditional views of others, that the diaphragm, by rising, mechanically compressed the cavities and the lower lesions adjacent to the diaphragm only. As with our experience with pneumothorax, sooner or later, upper lobe and apical cavitation had uncontrollable hemorrhages. Collapse with pneumothorax could not be obtained and, as a last resort, phrenicectomy was

tried. In one of the first cases, a chronic bleeder, the hemorrhage was checked. Soon after, the cough was diminished, the patient began gaining weight, sputum was diminished, and the patient, for the first time in two years, made rapid improvement Sometime later, an x-ray was taken and, to our surprise, the apical cavity was almost closed, parenchymal lesions were improved, and an infirmary case of three years became ambulant and returned to his family. We now believe that the entire lung shares in the process of relaxation, and, while it is true that lower lobe lesions usually are favorable cases for good results, upper lobe lesions, and even apical cavities, make wonderful improvement with this form of therapy. Of course, as in all forms of collapse therapy, it is the thin-walled cavities, centrally located, that yield best. Phrenicectomy has demonstrated the principle of partial collapse therapy. No one who has followed any large group of these cases and observed these cavity closures after hemidiaphragmatic paralysis, in which, it is said, the lung is collapsed only 25 per cent, can doubt the principle of selective collapse.

We study our cases carefully, especially with fluoroscope and films, to determine the mobility of diaphragm. In the presence of adhesions preventing the elevation of diaphragm, of course, we cannot expect as good results. However, I could show you several remarkable cases of cavity closure where the operation has been done in spite of massive adhesions and little movement of diaphragm. It appears to me that the improvements often seen in this type of case, with the added immobilization of chest and diaphragm, justify the operation in spite of the clinician's preconceived ideas of adhesions. Here, as in pneumothorax, the patient is often entitled to the attempt, especially in a simple operation without much hazard. You know, we humans are in the habit of figuring out the mechanisms of the functions of the human body in terms of our experience with inanimate mechanisms. We have attempted to apply these same mechanical principles to collapse therapy. We forget that we are dealing with a complicated human physiology about which our finite minds still know little.

In review of the subject, I find some surgeons recommend phrenic exeresis only as adjunct to other collapse therapy. I cannot agree with this view. I believe it constitutes one of the most valuable independent measures in the program for far-advanced tuberculosis. It is the experience of our clinic that cough and expectoration are diminished, many cavities are closed and that remarkable improvement frequently follows this procedure alone; however, it is also used in connection with pnemothorax therapy and as a preliminary to thoracoplasty. We find that in old, long-continued pneumothorax, phrenic evulsion is often of value in diminishing the frequency of refills and the amount of air necessary to maintain collapse.

We have, at the present time, one case under treatment in which we have induced partial pneumothorax of one lung and hemidiaphragmatic paralysis on the other side. We use it on practically all cases in which we cannot obtain pneumothorax. As in pneumothorax, we cannot always demand a good contralateral lung, and we have had the same experience as in pneumothorax, that the contralateral lung, although diseased, often shows wonderful improvement and even contracture of cavities.

With the years of added experience, thoracoplasty is now recognized as a standard procedure and is now not attended by the fear and dread of danger as in former years. In cases with far-advanced lesions and cavities, in which there is an adequate contralateral lung, and where pneumothorax and phrenic exeresis have failed to give results, then a more radical operation must be resorted to, either a partial or upper-stage thoracoplasty, if the lesion is confined to the apex, or a complete thoracoplasty where more extensive lesions and cavities are present.

Many of our patients are now enjoying comparatively good health and even usefulness since receiving thoracoplastic collapse. It is my opinion that many thoracic surgeons are still adopting a too conservative program and adhering to too strict indications. Where a patient has large cavities, is in danger of hemorrhage, extension of lesion, mixed infection, amyloidosis, spread of lesions, and where he has met failure with prolonged rigid rest therapy, and where pneumothorax and phrenic exeresis cannot obtain results, and if he still has an adequate contralateral lung, it is my opinion that he should be given the opportunity for a more radical collapse.

I am pleased to observe that more surgeons are adopting a more liberal program and are saving a great many patients who otherwise are doomed to early death.

When one has a hospital filled with these far-advanced infirmary cases who have met failure, he is forced to the realization that he must often do something more than sit passively by and give advice as to the value of hygienic treatment.

Since becoming alive to the greater possibilities of collapse therapy, the morale of both patient and personnel has changed. Now, an atmosphere of hope

prevails. A few years ago I had difficulty in "selling" a patient any form of collapse therapy. Most of them had been well coached as to the dangers and contra-indications of such therapy. Now, I can scarcely make ward rounds without one or more patients calling to me and wanting to know, "How about a pneumothorax in my case, Doctor?" "Don't you think I am a suitable case for a phrenic nerve operation?" "Doctor, don't you think I could stand a thoracoplasty?"

We now make a study of each case for possible collapse therapy, and we are surprised at the number who are suitable when a systematic study is made. There are so many possibilities with unilateral or bilateral pneumothorax, phrenicectomy alone or in combination with pneumothorax and thoracoplasty, that, once one really becomes interested, there is no end to the possibilities for saving lives.

Recently, I was talking with one of the leading thoracic surgeons in the United States, on collapse therapy. He told me of a tuberculosis hospital near his city, and with which he was associated, which admitted only the far-advanced, adult type of tuberculosis. He asked me to guess the percentage of patients in that hospital which were under some form of collapse therapy. Knowing his enthusiasm, I aimed high and guessed twenty-five per cent. He laughingly replied, "Doctor, I am afraid to tell you, I am afraid you cannot stand the shock." He then told me that seventy per cent of those patients had received, or were receiving, some form of collapse therapy, and that results obtained justified the procedure. It will be a long time before most of us reach such a stage of enthusiasm, but this example serves to show the future trend in collapse therapy.

A few days later I had the privilege of talking, on the same subject, with the chief of the medical service of one of the largest institutions in the United States caring for the far-advanced tuberculosis cases. This man is not a surgeon but an internist. He informed me that, since introducing collapse therapy into his institution, more and more the scope of this form of surgery was increasing and that, more and more, there was a tendency to depart from the old strict rules for indications for collapse therapy. It was his opinion that these doomed patients were entitled to some last resort chance for recovery, or, at least, to partial recovery.

It is my opinion that the percentage of patients receiving some form of collapse therapy is far too low in nearly all tuberculosis hospitals. I believe we are entering a new era in which most of us have only begun to realize the possibilities of future success.

SCALENIOTOMY

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(Read before the Eighteenth Annual Meeting of the Medical and Surgical Association of the Southwest, held at Albuquerque, N. M., Dec. 8-10, 1932.)

Since rest is the accepted treatment for all forms of tuberculosis, it follows that any measure designed to limit the movement of the lung and establish pulmonary rest mechanically, is entitled to consideration in the treatment of pulmonary tuberculosis. Its practicability is to be determined by the results obtained.

Scaleniotomy is a division of the fibers of the scaleni muscles. You will recall that there are four muscles in the scaleni group, and that they arise from the transverse processes of the third, fourth, fifth, sixth, and seventh cervical vertebrae, and insert into the first and second ribs. They are muscles of respiration, in that they elevate the first three ribs and, thereby, support them, opposing the action of the intercostal group.

PROCEDURE

The night before the operation, the patient is given three grains of sodium amytal by mouth, and in the morning, one hour before going to the operating room, is given six grains of sodium amytal by mouth and one two-hundredths of a grain of scopolamine hypodermically. He lies on the operating table in a supine position, with a small pillow under the shoulder of the side to be operated upon, and the head turned as far as possible in the opposite direction. The skin is prepared in the usual way, and a two per cent solution of novocaine is used to anesthetize the line of incision, which is placed two and a half centimeters above the clavicle and extends backward from the posterior edge of the sternocleidomastoid muscle about six centimeters, paralleling the clavicle.

The platysma muscle and superficial fascia are infiltrated through the skin. The skin is then incised, and the superficial tissues are separated by blunt dissection, when the scaleni can usually be felt just behind the posterior border of the cleidomastoid muscle. The deep cervical fascia covering the scaleni is separated, in a line paralleling the sternocleidomastoid muscle, at right angles to the skin incision. There is usually a pad of cervical fat covering the scalenus anticus, which is carefully separated from the sheath of the muscle. The phrenic nerve is generally found lying under the sheath and upon the muscle. The sheath and muscle fibers are anesthetized with one-half of one per cent novocaine solution, and by blunt

dissection the sheath is separated from the muscle fibers, as far down near their insertion into the first rib as it is possible to go, care being used to avoid the subclavian artery, which lies between the anticus and medius, upon the first rib. A grooved director, bent into the form of a hook, is slipped inside the sheath, encircling the muscle fibers, and the fibers are divided by a cautery knife. The scalenus medius is smaller, less important, but much more difficult to approach than the anticus, because it lies deeper and external to it, and is covered by some of the cords of the cervical plexus, which sometimes lie within its sheath. The cords are isolated and gently drawn inward, and the sheath and muscle fibers are anesthetized. The sheath is separated from the muscle fibers, proceeding downward to their tendinous insertion into the second rib, where, as before, the hooked grooved director is placed inside the sheath, under the muscle fibers, which are then divided by the cautery knife. No attempt is made to isolate the scalenus posticus or minimus separately, since they usually lie in the same sheath as the medius and are ordinarily indistinguishable from it. After complete hemostasis has been assured, the wound is closed in two layers, plain catgut being used in the fascia and Michel's clamps in the skin. No attempt is made to obliterate the cavity, ordinarily about the size of a large walnut, which results from the retraction of the severed edges of the muscle segments. The pad of fat that is encountered lying upon the sheath of the anticus is stitched down to the posterior sheath of the muscle, to prevent bridging of the muscle fibers.

DISCUSSION

The severing of the scaleni muscles allows the first two ribs to sag, and to a certain extent decreases the respiratory excursion of the first three ribs, thereby inducing a degree of rest in the region of the apex of the lung.

We have all had cases in which phrenic exeresis was done, that were not completely successful; and the reason for this was, presumably, that the immobilizing effect of the operation did not extend to the top of the lung. Indeed, in some cases of this sort, the respiratory activity of the upper part of the chest has been actually observed to be increased, as if to compensate for the immobilization at the base. It is in precisely these cases that the apical rest provided by scaleniotomy would be expected to be beneficial.

At operation for scaleniotomy in cases where the phrenic nerve has been previously evulsed, the scaleni were observed to be hypertrophied, presumably as a result of this compensatory increase of ventilation of the upper chest. In the cases where scaleniotomy was done, associated with phrenic exeresis, this increased movement of the upper chest was not observed.

In all types of operations done to immobilize the chest, unfortunate complications are a possibility; but, so far in this series of scaleniotomies, no complications have occurred, nor deformities resulted.

In this series there are sixty-six cases of scaleniotomy, some of which have been reported in a previous paper. In twenty-two of these, the phrenic nerve has been severed from six months to three years previously, and the resulting improvement had come to a standstill. Scaleniotomy resulted in further improvement in twelve of these twenty-two cases. There were three cases of tuberculosis, chiefly apical, associated with asthma, and, because it was believed that full action of the diaphragm was indispensable to respiration in this complication, the phrenic nerve was not cut, and scaleniotomy alone was done. There was clinical improvement in two of these three cases.

The combined operation was done in forty-one cases, with resulting improvement in twenty-five cases.

In two of these cases, pneumothorax had been established for a year or more, but with no improvement because of broad apical adhesions, and both of these cases were improved by the combined operation.

CONCLUSIONS

First: In cases with apical lesions, scaleniotomy should be combined with phrenic exeresis.

Second: When pneumothorax fails to collapse the upper part of the lung, because of broad adhesions at or near the apex, scaleniotomy should be done.

Third: When phrenic exeresis is contra-indicated, scaleniotomy alone should be tried in apical cases.

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There is being widely distributed an announcement of the Illinois College of Physicians and Surgeons, 20 North Ashland Boulevard, Chicago, which includes the following statement:

"Courses offered and requirements for graduation are class 'A' requirements."

Inasmuch as the Council on Medical Education and Hospitals of the American Medical Association is the only body which has ever rated medical schools as class A, it is clearly implied that the above named school conforms to the standards prescribed by this Council. Such an inference, however is wholly unwarranted. The above institution is conducted by a group of chiropractors and does not even remotely approach the standards of a class A medical school.

THE CLASSIFICATION OF NEPHRITIS FROM THE PATHO-PHYSIOLOGICAL VIEWPOINT

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(Read before the Eighteenth Annual Meeting of the Medical and Surgical Association of the Southwest, held at Albuquerque, New Mexico, Dec. 8-10, 1932.)

Nephritis, or Bright's disease, is a group of maladies in which certain non-suppurative lesions of the kidney are present.

The classification of nephritis is a source of great confusion to most clinicians, and various classifications have been given by them. There are many reasons why a satisfactory classification is difficult to attain. For the clinician, the greatest help in diagnosis and guidance to treatment is obtained when every case is analyzed so that the following points are considered independently:

1. Pathology of the kidney and other tissues.
2. Renal function.
3. Edema.
4. Blood pressure.
5. Anemia.
6. Uremia.

The anatomical structure of the kidney is familiar to everyone. There are three main constituents: the glomeruli, the tubules, and the blood vessels. This trinity of structure corresponds to the three main divisions of Bright's disease; namely:

1. Glomerular nephritis (primary involvement of the glomeruli).

2. Tubular nephritis (primary involvement of the tubules, or nephrosis).

3. Arterial nephritis (primary involvement of the arteries and arterioles, viz. arteriosclerotic nephritis or nephrosclerosis.

The glomerulus with its corresponding tubule, forms one structure and functional unit—the nephron—which can be compared to the neuron, the unit of the nervous system. An injury to a nerve cell is apt to be followed by changes in the nerve fiber, so injury to a glomerulus is reflected by changes in its tubule. The reasons for this are: (1). The greater part of the blood supply to the tubule first passes through the glomerulus, leaving it by the efferent arteriole. (2). When the glomerulus ceases to function, the tubule will atrophy from disuse. Also, the glomerulus will degenerate when its tubule is obstructed, as is observed in hydronephrosis. These changes in the glomeruli and tubules, with the resultant atrophy, lead to an increase of connective-tissue-replacement fibrosis and scar formation. Slowly developing reduction of arterial blood supply by disease of the arteries and arterioles and chronic venous hyperemia, results in disturbed nutrition of the glomeruli and tubular epithelium, and this results in all grades of atrophy to complete disappearance of these structures and replacement fibrosis (Simonds[1]).

The kidney has a reserve power comparable to the heart. Richards[2] made the important discovery that the number of active glomeruli, those through which the blood was circulating, varied greatly. Also the number of capillary loops within a single glomerulus at any given time may be only a fraction of the total number. This observation explains two features of renal pathology; namely, how a large number of glomeruli may be thrown out of action without any evidence of renal insufficiency, and also how a toxin acting diffusely but temporarily on the kidney may affect some glomeruli while sparing others.

In describing the pathology of nephritis it has been customary to consider only the anatomical changes in the kidney. The occurrence of protein, red blood cells, and casts in the urine is linked so closely with the renal lesion that it clarifies the morphological diagnosis to consider them separately.

By albuminuria is meant the presence of any protein material, not only albumin, in the urine. Besides albumin, globulin and other proteins are found in the urine. A normal urine is considered usually to be protein-free, indicating that the glomerular capillaries are impervious to the serum proteins. The quantity of protein eliminated in the urine varies greatly. There may be traces or the quantity may be large.

The daily fluctuations in chronic Bright's disease are of little significance. In acute Bright's a healing of the inflammatory process is accompanied by a diminution and disappearance of the albuminuria. The degree of the albuminuria is not an indication of the degree of the impairment of renal function, but is a sign of the type of Bright's disease which prevails. In acute and chronic glomerulonephritis and in nephrosis the albuminuria is marked, but in sclerotic disease associated with arteriosclerosis usually it is slight, may even be absent, although the kidney may be extensively involved and its secretory power reduced to the vanishing point.

Urine casts are microscopic structures which can be traced from the glomerulus. They are usually

associated with albuminuria. It is best to examine fresh samples of urine for casts, as they are easily dissolved on standing. This is true of dilute or alkaline specimens (Addis[3]). Hyaline casts are the most frequent and are characteristic of nephrosclerosis. Blood casts are usually pathognomonic of glomerular nephritis, usually in the acute stage. In cases of "lipoid nephrosis" double refractile lipoids have been found in the urine and embedded in the casts. Waxy casts were formerly characteristic of amyloid disease, but this idea has been discarded. They are usually found in chronic nephritis in which there is a degenerative process of the kidneys.

An occasional red blood cell is of no significance. Glomerular nephritis is characterized by blood in the urine. When the amount of blood in the urine is large, blood casts may occur. These casts always indicate glomerular nephritis, whether acute or chronic. With the subsidence of inflammatory process in the kidney, the hematuria has a tendency to diminish. Tubular nephritis (nephrosis) is characterized by an absence of red blood cells in the urine—when present here it is an indication that the glomeruli are also involved. In arteriosclerotic nephritis, red blood cells are absent. This is to be expected, since the circulation in the glomerular capillaries must diminish as the renal arterioles become sclerosed. When heart failure brings about passive congestion in such kidneys, red blood cells may occur in moderate number in the urine.

The pathological physiology of the accumulation of fluid in the subcutaneous tissues and the body cavities, the peritoneum, the pleura and the pericardium, has not been explained completely. Doubtless there are at least two distinctive types of edema. That occurring in diffuse glomerular nephritis, and that found in the edematous tubular nephritis (lipoid nephrosis). The two may be found combined in the same case, as in "glomerular nephritis with a nephrotic component." From the clinical aspect, two points of diagnostic value in regard to the edema of Bright's disease should be mentioned. In the first place, you will find that, when the body cavities are subject to distention in Bright's disease, it is the peritoneal space that is the site of election for edematous accumulation. The frequency of infection of this peritoneal fluid in edematous tubular nephritis, suggests a greatly depressed functional activity and vulnerability in the lymphatic and vascular apparatus supplying the peritoneum: when anasarca is the result of cardiac failure the pleura inevitably shows the first accumulation of fluid and then the peritoneum follows. This predominance of ascites over hydrothorax, or vice versa, furnishes valuable information in diagnosis of the heart and of Bright's disease in cases where both are factors.

In the second place, there is the well-known fact that the edema of Bright's disease is not seen first in the dependent parts, as in passive congestion, but is prone to make its appearance about the face. This phenomenon and the fact that the edema frequently precedes the advent of the albuminuria, indicate that the fluid retention in Bright's disease is not entirely due to the inability of the kidney to excrete water, but is associated with physical and chemical disturbances in the blood, the lymph, the capillaries, and the soft tissues. The edema formation depends upon, first, depletion of the serum albumin of the blood by its loss in the urine, and, second, the retention of crystalloids in the tissues. Barker and Kirk[4] have shown by experiments upon dogs and by quantitative studies of serum albumin of the blood of nephrotic patients, that the reduction of the serum albumin of the blood to 1 per cent or below is followed by the accumulation of fluid in the subcutaneous tissues (edema). Fahr[5] found that the normal values for the osmotic pressure of the plasma proteins were from 20 to 23 mg. of mercury. The retention of crystalloids in the tissues is apparently not due to inability of the kidneys to excrete them, but due to some other mechanism, possibly concerned with an effort to maintain the concentration of the proteins of the blood at their normal level. The accumulation of chlorides in the tissues increases the osmotic pressure of the tissue fluids, and this, in turn, leads to withdrawal of water from the blood, causing concentration of serum albumin in the blood and increasing the fluid content of the tissues, inducing edema. Speaking in general terms, we can say, at least in chronic forms of nephritis, the edema varies inversely as the blood pressure; as the blood pressure rises, the edema tends to disappear. This is merely a clinical observation, so we are not justified in stating that there is any constant relationship between hypertension and the disappearance of edema. Hypertension is one of the most characteristic phenomena of chronic glomerulonephritis. It is seen in about one-third of the acute cases. Here we are dealing with a beneficent compensatory reaction. As Ludwig long ago pointed out, the filtration of urine through the glomerulus is a physical process in which the most important factor is the pressure of the circulating blood. If that process of filtration is rendered more difficult, as by partial or complete occlusion of large numbers of glomeruli, the simplest method of overcoming the difficulty is by a raising

of the blood pressure brought about by hypertrophy of the left ventricle. Up to a certain point this rise of pressure will enable the kidney to perform its normal function—if anything interferes with the rise of pressure, the kidney will at once suffer.

A rise in the blood urea which may be taken to indicate a retention of nonprotein nitrogen in general, must be attributed to some defect of the glomerular filtration apparatus. A raised blood urea in itself, unless of very severe grade, need not have great significance, for it may be due to extra-renal causes, and may be associated with perfectly efficient kidneys. The relation between blood urea concentration and the amount of functioning renal tissue has been investigated by McKay[6], and he found that, as long as 50 per cent or more of the kidney tissue is functioning, the urea in the blood is not over 18 mg. per 100 cc. which is within normal limits. This percentage also applies to the phenolphthalein test for, as long as we have 50 per cent of normal kidney tissue functioning, we find no deficient percentage of phthalein output.

Osler defines uremia as a toxemia developing in the course of nephritis or in conditions associated with anuria. The exact nature of the toxemia and the origin of the toxin which gives rise to it, are not known. Until recently, urea was considered a toxic substance. This we know is not true. There may be the highest concentration of urea in the blood, without any uremia. Many different forms of uremia, such as visceral uremia, cardiac uremia, asthmatic uremia, have been described. When all of them are considered, it seems that there are three forms of uremia which merit consideration, namely: (1) uremia of cerebral arteriosclerosis; (2) uremia of acute diffuse glomerulonephritis; (3) uremia of renal insufficiency, or retention uremia.

The uremia of cerebral arteriosclerosis may develop as a sequel to a prolonged hypertension whenever this occurs in Bright's disease. This is usual in the course of essential hypertension, primary contracted kidney, chronic diffuse glomerulonephritis, or secondary contracted kidney. The uremia of acute diffuse glomerulonephritis accompanies acute diffuse glomerulonephrosis or an acute exacerbation occurring in the course of chronic nephritis. The uremia of renal insufficiency, or better called retention uremia, is the sequel of a prolonged and marked degree of diminished excretory activity of the kidneys from any cause whatever. The forms of Bright's disease in which it occurs are the subacute diffuse glomerulonephritis, the primary and secondary contracted kidneys, and the anuric form of tubular nephritis (bichloride of mercury poisoning). The blood pressure of the onset of retention uremia is characteristic; that is, it is usually elevated. As the condition becomes more serious and the fatal termination approaches, the blood pressure frequently drops. Both the systolic and diastolic pressures are found to be lower at this time. The cause of this lowering of the blood pressure level is probably due to the weakening of the heart and the arteries as well. As a rule, death may be expected within two to four weeks after the blood pressure is lowered.

It is not an uncommon experience to find that, in the uremia accompanying acute diffuse glomerular nephritis, or acute exacerbations of chronic nephritis, there are no large increases in the blood urea, uric acid, and so forth. In other words, renal insufficiency and the retention of urinary excretory products are, contrary to the prevalent belief, not the only factors present in the evolution of uremia. Contributory factors may be (1) edema of the brain, (2) angiospasm of the cerebral vessels, (3) damage to the brain by the hemolytic streptococcus toxin which also caused the nephritis.

The intestines may be the seat of the so-called uremic ulcers.. These may occur anywhere in the digestive tract, from the pharynx to the rectum. They are often responsible for the presence of considerable amounts of blood in the vomitus and the stools. The kidneys and urine are the keystone upon which the advent and progress of the uremia of renal insufficiency depend. The progressively diminished activity of the kidneys is noted first when the blood chemistry is maintained at a normal level through polyuria. When this compensatory process fails, the blood urea and other substances begin to accumulate and give warning of impending danger. There are two influences that may accelerate the course of events by influencing renal activity adversely. These are cardiac insufficiency and an acute exacerbation of a chronic nephritis. Albumin may be present in large amounts or as mere traces in the urine. This depends upon the lesion in the kidneys and, in itself, has no bearing upon the adequacy of renal function.

The volume of urine becomes progressively and inevitably less; in spite of sufficient fluid intake, the urinary secretion may cease entirely. It is this development of oliguria that is responsible for the curtailment of kidney function and the fatal termination of the uremia of renal insufficiency . The specific gravity of the urine is constantly fixed around 1010. At the same time there is no denying the fact that uremia is almost always associated with

marked urea retention, not only in the blood, but also in the tissues.

One of the constant symptoms of nephritis is anemia, evidenced by the increasing pallor of the patient. This anemia is undoubtedly of secondary type. By making hemoglobin estimates, the progress of the case may often be adjudged, as well as by the routine non-protein nitrogen determinations. The severity of anemia is directly proportioned to the degree of impairment of renal function. I cannot but believe that an important cause of the anemia has been due to the low protein diets that these patients were put upon prior to the present time, when one no longer hesitates to maintain diets of fairly high protein intake, and with no more appreciable damage to the diseased kidney than formerly when the protein molecule was forbidden. A safe formula in the average adult is about 70 gm. of protein plus the amount of protein that is lost in the urine. This procedure also may overcome a possible renal insufficiency by the conversion of the protein unto urea, which is itself one of the best diuretics known.

In brief, I believe it is timely to stress the important point that the first indication of renal functional impairment is the diminution in the kidneys to eliminate the solid constituents of the urine in their normal concentration. This can be very simply determined by making routine specific gravity readings. The normal kidneys possess the power of elimination of solids within wide limits—regardless of the amount of water available for their solution. Consequently, urine of low volume has a high specific gravity; that of large volume, a low specific gravity. When renal function is impaired, the ability of the kidneys to elaborate a concentrated urine is diminished; with increasing renal damage the kidney loses its ability, not only to concentrate, but also to dilute urine.

One of the simplest methods available for the interpretation of renal function, is the information gained by running routine Mosenthal two-hour specific-gravity test. It yields more data than any other test now in vogue to enable the clinician to interpret renal activity on a rational basis of physiologic efficiency or impairment.

In conclusion, it seems evident that, by merely classifying nephritis by name or group, a false sense of security is fostered. Therefore, it is imperative that all of the factors that contribute to the evolution of Bright's disease must be evaluated before a rational form of therapy can be advised.

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PLACENTA ACCRETA

Report of Case with Review of Literature

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(Read before Pima County Medical Society, September 13, 1932.)

"Placenta accreta is a rare complication of pregnancy in which there is a more or less complete absence of the decidua basalis, and in which the chorionic villi invade the musculature of the uterus.'"

"Placenta accreta, therefore, may be described as the implantation of the ovum and the development of the placenta on a uterine wall in which there is a total or almost total absence of the decidua serotina. The chorionic villi erode themselves into the muscle fibers, hence the uterine muscle partly degenerates, resulting in a thinning of the uterine wall over the placental site, even to the point of rupture. An appreciation of this pathology shows why the mortality from hemorrhage, sepsis, and perforation, makes this condition so formidable.'"

"Various estimates have been made of the frequency of this complication. Polak places the incidence of accreta at approximately one in 6000; B. C. Hirst, one in 40,000. Polak reports four cases met during an extensive practice covering thirty years. Of these, the first three were treated conservatively, by attempted manual removal, and terminated fatally as result of sepsis. The fourth case, a prmipara, seven months gravid, with a complicating large submumous fibroid, recovered following a supracervical hysterectomy. Frankl, in a discussion of this paper, could recall but six cases of true placenta accreta, of the immense material encountered in his clinic; and agreed with Polak that the indication is definite for extirpation of the uterus. Out of thirty-six cases collected by Kraul, only eight women recovered. A survey of the literature by me has revealed a total of for-

ty-one cases reported, in only twenty of which the placenta was examined in situ. Kraul describes three cases occurring in a total of 60,000 deliveries during the last twenty-three years at his clinic. Of these, the first two were fatal, the third survived a vaginal hysterectomy. Nathanson, in an excellent contribution to this subject, reports four cases occurring in a collective series of 75,000 deliveries, placing the incidence at approximately one in 20,000. Dietrich quotes the following authors, who, in reporting similar cases, stressed the excessively thinned-out condition of the uterine wall: Kwostansky-Meyer Ruegg (fundus, 1 mm. thin); Schwenderer (2 mm.); Bauereisen (3 mm.); E. Martin could palpate the intestines through the thinned-out uterine wall; Wegelin (1-3 mm. thin); R. Meyer (in placed, ½-1 mm.); Schmidt (in his first case, 4 mm., in his second, ¼ mm. thin); R. Meyer (in places, ½-1 mm.); the rupture occurred in the tenth month, and was diagnosed and operated upon two weeks later. Schwenderer describes a case in which Crede's maneuver led to a rupture of the uterine wall thinned out by placenta accreta.

"In the case of a para v, reported by Tennant, it was demonstrated that the placental attachment may not alone penetrate the peritoneal coat of the uterus, but may actually invade the visceral cavity. Schweitzer's case of placenta accreta was at the same time, a placenta previa cervicalis. Jaschke also speaks of the "burrowing" of the fetal elements into the cervical musculature in low placental insertions. Dietrich reports in detail the case of a forty-year-old para iv, with a very significant previous history in her four deliveries, including two manual removals of the placenta, one febrile puerperium, two severe postpartum hemorrhages, and two postpartum curettages. In the present delivery, the patient was admitted with mild labor pains, suddenly collapsed, and, following version and extraction, died. The autopsy showed a placenta accreta of the membranacea type, with a spontaneous perforation of the uterus, and partial extrusion of the placenta. He calls attention to the fact that a seven-year amenorrhea has existed between the preceding delivery and the case now reported, which he attributes to an atrophy of the mucosa due to an over-zealous curettage. This factor has likewise been stressed by other authors (Fritsch, Kustner, Veit, Wertheim), though the ability of the uterine mucosa to restitutio ad integrum, is often remarkable. Dietrich also describes a case of placenta accreta, reported by Baumgart-Benecke, due to an atrophied mucosa, following an amenorrhea of four years' duration (due to atmocausis); because of infection and scar changes, a Porro's hysterectomy was performed. Similar cases were reported by Holzapfel and Frankenstein, following vaporization therapy."

"When the placenta fails to be delivered, following the birth of the child, there are two conditions either of which may be present. The placenta, after separation from the uterine wall, may be retained in the birth canal. Such a case would show bleeding, descent of the cord and a round, firm fundus several centimeters higher than the position it had occupied immediately after the birth of the child. The other possibility is that of adherent placenta, in which condition separation from the uterine wall has not occurred. There would, therefore, be no bleeding from the placental site, no descent of the cord, and intermittent uterine contractions would still be felt.

"To the presence of an adherent placenta, the gloved hand, introduced into the uterus under careful aseptic precautions, may be able to demonstrate a line of cleavage, and so remove the products of conception. Such a situation might be found when the placenta is large and thinned out, or when implantation has occurred in a tubal corner. When no line of cleavage can be found, one may be dealing with a placenta accreta, an entirely different problem, which demands different treatment."

"The causes of this condition are obviously those which lead to destruction of the uterine mucosa: they would include repeated or too vigorous curettage, endometritis, submucous fibroid and previous manual removal. A history of any of these should make one prepared to deal with a placenta accreta."

CASE REPORT

Mrs. C. D., aged thirty-one, first seen by me September 30, 1931, at which time she was six months pregnant. In 1928 she had been curetted. In September, 1929, she was delivered by cesarean section. The indication for section, according to patient's statement, being a delayed onset of labor. The past and family history were negative.

The physical examination was essentially negative except for a few carious teeth, chronically infected tonsils, and the old postoperative suprapubic scar.

The estimated time of confinement was December 25, 1931. The patient was admitted to the hospital in labor December 30, at three a. m. Position L. O. A. At 1:45 a. m., the head was down on perineum. Episiotomy was done and the baby was delivered with low forceps. Repair of perineum was completed at 2:30. At 3 p. m. the uterus had not assumed the characteristic ball-like shape and position indicating the separation of the placenta, nor was there any descent of the cord, but there was a slight hemorrhage. After gently attempting to express the placenta by Crede's method, with failure, light ether anesthesia was administered. Under

strict aseptic precautions, the right hand was passed into the uterus and particular note was made of the fact that there was a complete absence of the line of cleavage by which the normal separation of the placenta is effected. With considerable difficulty a piece of placental tissue was dug out with the fingers, the major portion of the placenta being left in situ. Immediately, the patient showed signs of air hunger and collapse. I considered the possibility of placental accretion, and the uterus was packed with gauze at once, preparatory to a hysterectomy. The patient failed to response to stimulants, artificial respiration, and oxygen carbon dioxide inhalations. Death ensued within five minutes after onset of collapse, my impression at this time being that death was due to thrombosis and embolism, which was confirmed by autopsy.

At this time I was permitted to perform a regional autopsy only, with the understanding that no organs be removed. Through a midline suprapubic incision, the uterus was exposed. The fundus extended to the umbilicus. On the anterior surface of the uterus near the fundus, in the midline, incorporating a portion of the old cesarean scar, was an area about two inches in diameter where the myometrium was thinned to approximately 2 mm. in thickness. The uterus was incised and the gauze pack was removed. The thinned-out area marked the site where I had removed a portion of the placenta manually.

Several days elapsed before permission was obtained to exhume the body for a complete autopsy, which was performed January 14, 1932.

The following is the report of Dr. Robert Alan Hicks, pathologist, who has given me much valuable assistance in reporting this case.

PATHOLOGIST'S REPORT

The features of this case which make it somewhat distinctive from the instances of placenta accreta already reported are, first, possible differences in the etiology of this case and the etiology hitherto reported. Second, the unusual histological picture of the relative absence of the large vessels normally found in the spongy layer of the decidua basalis.

We may well begin with the picture of the gross specimen as we saw it at autopsy. There was no evidence of inflammatory reaction in the peritoneum. The corpus uteri measured twenty-one centimeters from the dome of the fundus to tne cervix, fifteen centimeters from horn to horn, and was collapsed on itself antero-posteriorly to a thickness of about five centimeters. As the uterus was opened in the anterior midline, we first observed the universal implantation of the placenta. There was no point at which the edge of the placenta could be differentiated and on gross section it was not possible to determine the point at which myometrium left off and placental tissue began. It was further impossible to establish any line of cleavage between the two structures.

Microscopic sections were prepared from the dome of the fundus and at all levels down the corpus uteri to the cervix. It was found that, at the dome of the fundus, the musculature was thinned to an actual thickness of two to three millimeters and that at this point the decidua compacta and the decidual spongiosa were entirely lacking. The choronic villi extended to and sometimes penetrated the fibers of the myometrium. The large vessels found ordinarily in the decidua spongiosa were entirely lacking. This feature is of great significance because engorgement of these vessels after the delivery of the fetus is a major factor in the normal separation of the placenta. The structures present here were amorphous fibrinous masses, sometimes canalized, and many large stellate cells of the fetal ectoderm or trophoblast. In previous reports of the histopathology of placenta accreta, these structures have been interpreted as part of the picture of accretion, but we know that they are present normally in the line of separation of normal placenta and decidua spongiosa.

In sections prepared down the wall of the corpus toward the cervix, the only essential change in the picture was a progressive thickening of the myometrium, and an occasional meager development of the decidua compacta and spongiosa. However, throughout the extent of the atrophic and insufficient spongy decidua present, we never found a trace of glandular structure. The complete absence of both the endometrial glands and the loose associated endometrial stroma, is sufficient to establish the diagnosis of placenta accreta, and this was the finding in the wall of the corpus. In addition to this, at the dome of the fundus we found the marked thinning of the myometrium so typical of placenta accreta and, finally, the infiltration of the myometrium by the chorionic villi. This emphasis on the histopathology of placenta accreta at the dome of the fundus and in the midline, in contrast to the less typical finding farther down the side wall of the fundus, makes us inclined to associate the development of the placenta accreta with the implantation of the ovum in the dome along the line of the old cesarean scar. This cannot be an absolute conclusion, however, as we know that the endometrium over the dome of the fundus and at the angles is always less redundant than it is in the other portions of the uterine cavity. Such normal hypoplasia of the endometrium, with the addition of injury by cesarean section and currettage, constitutes the etiology of this case.

Large fresh venous thrombi found as emboli in the pulmonary arteries, constituted the immediate cause of death.

(Slides of the microscopic pathology of this tissue may be obtained from the authors)

CONCLUSIONS

1. Placenta accreta is a rare complication of pregnancy and there have been reported only forty-two cases, including this one.

2. The characteristic features of this abnormality

are partial or total absence of the decidua basalis and an invasion of the musculature of the uterus by chorionic villi.

3. In addition to repeated or too vigorous curettage, endometritis, submucous fibroid, and previous manual removal, as etiological factors, this case adds the possible significance of previous cesarean section. It must be born in mind also that the manual removal of the placenta at the time of cesarean section would further increase the possibility of a hypoplasia of the endometrium, with a resulting tendency to placenta accreta.

4. In reviewing literature, we are warned of the following dangers: perforation and rupture of the uterus, hemorrhage, shock, and sepsis. This case adds the danger of embolism from venous thrombosis.

5. In the presence of an attached placenta with no descent of the cord and without bleeding, aseptic exploration under anesthesia should be made to determine the subsequent procedure. If no line of cleavage can be demonstrated, hysterectomy should be done.

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CONVERGENT SQUINT, ITS CAUSE AND TREATMENT

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(Read before the Maricopa County Medical Society, Nov. 14, 1932.)

The consequences of a neglected or improperly treated squint, both in childhood and in later life are sufficiently serious and important to make it worth while briefly to mention them and to bring to your attention what is now the commonly accepted cause of internal squint, or strabismus, and its treatment. I shall discuss only concomitant or non-paralytic squint of the internal variety, because it is much more common than external squint and the cause and treatment is somewhat different.

A cross-eyed child very quickly, after starting to school, or perhaps before, becomes conscious of the deformity by constant reminders of its playmates, becomes shy and bashful and may quickly develop an inferiority complex. If the deformity remains until adolescence, its effect on the individual's social career is apt to be disastrous. This is, of course, true if it be a girl. It also lessens the chances of finding a gainful occupation. Occasionally an eye which crossed in childhood, will spontaneously straighten, or nearly so, as the age of puberty is reached. In this event, the eye is practically blind and its possessor has passed the age at which good vision could have been developed. He is then deprived of the opportunity of a career in a multitude of directions, such as the military services, railroads, steamships, factories which employ machinery, and in most lines of endeavor where some hazard exists and a rigid physical examination is required.

Why do children squint? There are several factors which enter into its causation. The theory of Claude Worth, of London, is the most plausible and is accepted by most observers. This explanation leads to a rational treatment which is in the main successful.

When a person with straight eyes gazes at an object, an image of that object is simultaneously formed on the macula of each retina. The object being seen from a slightly different angle with each eye, the images are slightly different and, when fused into one image by the brain, become a stereoscopic picture with a sense of depth. This is called fusion. This fusion sense begins developing at about the sixth month and is fully developed by the sixth or seventh year. According to Worth, if it is not developed by that time, it will not develop. It is this desire to see an object stereoscopically, thus getting a sense of depth as well as avoiding troublesome double vision, that is responsible for the eyes remaining parallel in spite of overactive, weak, or otherwise poorly balanced muscles. It is this fusion sense that, though a muscle may be weak or placed too far back on the globe, will cause an extra amount of nervous energy to be supplied to the muscle, in order to overcome its deficiency and to hold the eye parallel with the opposite eye. As a proof of this, we may take a person with straight eyes and normal vision and place a weak prism before one eye, thus displacing the object seen with this eye. If the fusion faculty is present, this eye will turn by reason of increased nervous energy in order to keep the image of the object on the macula and maintain binocular single vision. If now we increase the strength of the prism to the point where the eye can no longer do this, and produce double vision (diplopia), it will

be seen that, in a great proportion of individuals, there is a deviation of the eyes from the parallel, either in the vertical or horizontal direction, or both (heterophoria).

In internal strabismus, this fusion sense is either entirely absent or very weak. In the absence of it, there is nothing to keep the eyes parallel except a perfect balance of the muscles of the eye, and anything that might upset this balance will contribute to a squint. The vast majority of children with squint have a relatively high degree of hyperopia, combined frequently with astigmatism. Now, given a child with an absence of the fusion sense and having a high hyperopia, it is a simple matter for a squint to occur. The child, in order to see distant objects, is compelled to accommodate, that is, to increase the curvature of the lens, and because the accommodation and convergence are so closely linked together that one cannot occur without the other, there is a tendency for the eyes to converge when they should remain parallel. In looking at close objects there is, of course, more accommodation and a still greater amount of convergence, for near objects lead to double vision, and it is to avoid this troublesome double vision that the child unconsciously turns the eye inward in order better to suppress the poorer image. In the beginning, the deviation may be slight and periodic, straightening up when the eye is at rest. After a while, however, the squint becomes less periodic and more pronounced. The internal rectus muscle becomes contracted, and the opposite muscle, the external rectus, becomes attenuated and loses its tone. It is usual for one eye to have slightly more hyperopia and hyperopic astigmatism, therefore less vision, than its fellow, and it is this eye that eventually turns in. We thus have the mechanism of a constant squint.

Frequently a mother will state that the child's eyes were straight until it contracted whooping cough, measles, or some other disease, and thereafter the squint developed. According to Worth, these children probably had a very poor fusion sense, the attack being sufficient to alter the nervous energy supplying already poorly balanced muscles. Birth injuries, especially to the sixth nerve, improperly inserted muscles, macular lesions, and corneal scars, are occasionally responsible for a squint.

A small proportion of the internal squints, about 15 per cent, are alternating. That is, the right eye may turn in one moment and the left eye the next. These children usually have good vision in each eye and very little or no refractive error. They have no fusion sense and do not develop any, even with correct and prolonged treatment. They apparently have sufficient muscular or nervous incoordination to produce a diplopia and the eye is turned completely inward in order that one image may be completely ignored.

Now, we know that an eye which is turning inward will not develop vision, will quickly lose what vision it has, and that it will remain very nearly blind if not trained in early life. We also know that, if the fusion faculty has not developed by the sixth or seventh year, it will not develop. It follows, therefore, that these children should be treated without delay in order to correct these two conditions. This is not accomplished by the prescribing of glasses and forgetting them, nor is it accomplished by immediately resorting to an operation. The vision in each eye should be ascertained. The eyes should be thoroughly atropinized in order to unmask all latent hyperopia, and glasses should be prescribed if hyperopia or astigmatism is found. A child is never too young to wear glasses. Worth has prescribed them as early as the sixth month. Generally speaking, however, it is rarely necessary to resort to treatment at this age because a squint generally appears later, usually around the second year. At this age they very quickly become accustomed to glasses, realizing that they see better. Mothers may object to glasses in the belief that they might be a source of injury, but experience has shown that they are, on the contrary, a protection. The prescribing of glasses for a child does not necessarily mean that they will have to be worn permanently. Frequently, when the squinting eye has regained its vision and lost its deviation, if the refractive error is not of too high a degree, the glasses may be dispensed with. Quite often, when the excess accommodation is relieved by glasses, there is either a return to parallelism, or partially so. This is particularly true if the glasses are prescribed while the squint is still periodic.

If the eye continues to deviate, no time should be lost in taking measures to prevent the further loss of vision through disuse. This can be accomplished by putting a cover over the good eye for a week or two at a time, thus forcing the use of the squinting eye. After some vision has returned, atropine may be instilled daily into the good eye, thus paralyzing its accommodation and forcing the use of the squinting eye for near vision, and the normal eye for distant vision.

Just as soon as the vision has sufficiently improved, and if the child is old enough, stereoscopic exer-

cises should be started, in order to develop the fusion sense. If the deviation is of a low degree, the eye will become straight as fusion develops, and when it is fully developed, the eyes will remain straight even though the glasses are discarded.

If the deviation remains great, after a trial with glasses, a surgical correction is in order, to be followed up with stereoscopic exercises. I shall not go into the question of surgical procedures except to say that operative measures to correct an internal squint usually consist of shortening the external rectus muscle of the affected eye. If this falls short of a full correction, the externus of the normal eye may be shortened later.

The O'Connor cinch method of muscle shortening is, in my opinion, the most satisfactory method. For the past two or three years it has been employed exclusively in the eye services at the University of California and Stanford Medical Schools, with most satisfactory results.

Some of its advantages are: its simplicity; the absence of infection and sloughing, sometimes seen following a Reese resection; a more accurate estimate of the amount of correction to be achieved by reason of the number of dermol strands used.

To summarize, I should say that: 1) A squinting eye does not develop vision and loses the vision it once had. (2) It rarely straightens without treatment and, if it does straighten after the sixth or seventh year, the eye remains industrially blind. (3) Treatment in the very beginning is indicated, consisting of thorough refraction, glasses if necessary, training of vision, the developing of the fusion faculty and perhaps operation. (4) Surgical correction of squint after the sixth or seventh year will achieve only cosmetic result.

MUCOCELE AND PYOCELE OF THE FRONTAL SINUS

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(Read before the Maricopa County Medical Society, Nov. 14, 1932.)

Pyocele of the nasal accessory sinuses is an uncommon entity. About 200 cases have been reported, notably by Gerber, Dabney and Boenninghaus, while Beyer recently reported a case in explanation of the x-ray findings.

CASE REPORT

This patient, D. A., aged 13, was first seen October 6, 1930. She was complaining of very severe headaches and head pains, and presented a large conical swelling over the forehead, slightly to the left of the center, and accompanied by edema of the left eyelids. The conical swelling was tense and appeared to be a large abscess under considerable pressure. Palpation showed it to be definitely fluctuating.

The past history showed a long succession of illnesses. At birth she had a congenital heart defect which persisted, and she has always had a loud systolic murmur and a palpable thrill, while the heart has been considerably enlarged. She developed fairly well until the age of seven (1927), when she contracted bronchial pneumonia and, as a complication, had a pneumococcic mastoiditis on the right associated with a sinus thrombosis. A simple mastoidectomy with ligation of the corresponding jugular and exposure and emptying of the lateral sinus, was done. This was followed within a year (1928) by a radical mastoidectomy on the right. One year later (1929) an acute otitis media appeared on the left and following paracentesis this ear drained for five months. During this period she developed an acute endocarditis and was desperately ill for weeks. There was some improvement to February, 1930, when all her nasal accessory sinuses became infected subsequent to a cold, and at this time it was first noted that there was some swelling over the forehead. This persisted for about ten days and subsided when there was a sudden gush of pus into the left nostril. Similar attacks recurred at about monthly intervals, with increasing severity, and in October the patient was advised to leave the rigorous climate of New York and enlist the aid of Arizona's sunshine. Just before leaving New York, the swelling again appeared over the forehead, but on the way out the mother noted that its character had changed somewhat, in that it seemed more localized. In this condition she presented herself for treatment.

The child was obviously ill and in great pain. There was a temperature ranging to 100°. The mass over the forehead was excruciatingly tender. There was a purulent discharge from both antral areas and on the left a slight discharge from the frontal sinus area. It was impossible to enter the naso-frontal duct with a probe. No eye symptoms were present. The patient's weight was 60 pounds. X-ray findings were: "Confirming verbal report, we find abnormal density in antrum of the left side. Frontal sinuses do not show gross abnormality in shadow. Transillumination of sinuses shows both antra and both frontals hazy."

The diagnosis was bilateral maxillary sinusitis, pyocele of the left frontal sinus, with erosion of the anterior sinus plate. Any procedure except intranasal tamponing was emphatically denied and it was only after several days, during which time the condition showed no evidence of abating, that a small incision was allowed for drainage. Following this incision and the evacuation of a large amount of pus, the patient immediately improved. One month later an intranasal enlargement of the naso-

frontal duct established adequate drainage, and the external discharge diminished markedly. The condition now slowly improved over a long period of time. When the edema had subsided, a defect of the anterior frontal plate was found, which apparently involved most of the area and extended downward along the inner angle of the orbit. A permanent fistulous tract persisted into the superior border of the sinus approximately in the center of the forehead. Drainage stopped from this tract about one year ago.

At the present time there is no discharge, either externally or through the nose, and the patient is symptom free. Her weight has increased to about 100 pounds and she appears normal for her age and in excellent health. The bony defect is practically erased.

COMMENT

Following an acute inflammation subsequent to a head cold, this patient developed a purulent frontal sinusitis with periodic occlusion of the naso-frontal duct. Finally, an erosion of the anterior plate of the frontal occurred and an abscess developed superficially. External and intranasal drainage cleared the condition. The end result has probably been a complete fibrosis of the left frontal sinus, with very slight deformity in the region of a persistent sinus.

Thet x-ray findings appear anomalous. Beyer explains this by the absorption of lime salts and consequent loss of density, giving the appearance of transparency. In most cases the x-ray is reported as being of less aid than it usually is in sinus involvement.

This case showed no orbital involvement, although this appears in about 75 per cent of cases and is usually associated with exophthalmus.

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THE SAFEST MILK SUPPLY

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The term "safe" when applied to milk is of necessity a relative term. Theoretically, milk which had been properly pasteurized and then handled in such a manner that no pathogenic organisms were permitted to enter it after pasteurization, would be safe. Actually, such milk might not be safe. It has been demonstrated that, if certain strains of staphylococcus and streptococcus are permitted to develop in large numbers in milk, toxic substances are produced which are not destroyed at pasteurization temperatures and which are capable of producing intestinal disturbances similar to those produced by the living organisms, although no actual infection occurs. A number of years ago, Park stated that the intestinal disturbances of children in New York City apparently were not influenced materially by the types of bacteria found in the milk, but that there was a direct relation between the total bacterial count and the incidence of such complaints. So, if properly pasteurized milk was held at such temperature that the bacteria which survived pasteurization could multiply to sufficient numbers, they might produce intestinal disturbances in certain children. Pasteurization alone is not an absolute safeguard, although it is the best single safeguard known.

Certified milk is usually regarded as the nearest to the ideal for raw milk. However, there have been a few milk-borne epidemics from certified milk. It has been shown that a cow may begin eliminating streptococci or staphylococci in her milk at least six days before any noticeable changes occur in the udder or any physical changes occur in the appearance of the milk. If the organism in such cases should be pathogenic for man, many persons might become infected before the offending animal was discovered.

Perhaps we might go a step further and say that certified milk which has been properly pasteurized is the safest milk. I will agree that this would be ideal. However, for the present at least, it is entirely impracticable.

Do not think that I do not approve of pasteurization and of certified milk. I stated that pasteurization is the best single safety factor which we know how to apply to milk. Certified milk is the ideal for raw milk, but because of the cost of production, the quantity used will probably always be limited. However, the inspiration and leadership of certified milk has been of inestimable value to the cause of milk sanitation.

For some reason, a part of the American public does not like, or imagines that it does not like, pasteurized milk. There are some economic reasons against pasteurization in certain instances, but even more convincing arguments for it.

The universal production of certified milk is not practicable. Public taste or public sentiment renders

unlikely compulsory pasteurization of the entire milk supply for every community.

I have stated that no milk supply can be considered absolutely safe. However, we may say that the safest milk supply is one that has had every practicable sanitary and physical precaution thrown around it in its production and handling. These precautions should be reasonably effective and yet should be of such nature as not to increase the cost of the milk to the point which will necessitate lowered consumption. They should also be of such nature that the public can realize them, thus increasing confidence in the milk supply.

You will notice that, in my definition of the "safest" milk supply, I have stated that the sanitary regulations which are applied should be reasonably effective, economical, and of such nature as to tend to increase public confidence in the supply and, thus, to increase consumption. To be reasonably effective, they must be workable and cover all the recognized, essential milk-sanitation practices. To be economical, they should be of such nature that the average dairyman can afford the time and expense for their application. To increase public confidence, they should be of such nature that the public can be constantly informed of the extent of their application, and they should be uniform.

The U. S. Public Health Service has spent more than eight years in intensive study and research on the problem of providing the safest milk supplies for American cities and of unifying milk sanitation practices. They have sought a system which would be applicable to small as well as large cities and which could be used in every part of the country. Soon after the Public Health Service became actively interested in this subject, what was first called a model milk ordinance was drafted and recommended to a few cities. Later this was improved upon and recommended to a few cities under the title of the Standard Milk Ordinance. The name has now been changed to "The U. S. Public Health Service Milk Ordinance", but the plan is essentially the same as that which the Public Health Service has been recommending to state and city health departments for eight years.

During that interval, over 480 cities, located in 27 states and having a total population of over 8,000,000, have voluntarily adopted the program in a coordinated effort to improve and to unify milk sanitation methods.

The Public Health Service milk sanitation program may be summarized as follows:

(1) In order to prevent milk-borne disease outbreaks similar to the more than three hundred which have been reported in the United States in the past seven years, federal, state, and local health authorities have three major public health duties: (a) to promote the proper pasteurization of as large a percentage of the total milk supply as possible, (b) to promote the proper sanitary production of that part of the milk supply which is pasteurized, and (c) to promote the safeguarding of the remaining part of the milk supply as far as raw milk can practicably be safeguarded.

(2) These objectives will not be adequately realized nationally except through a nation-wide standardization of milk control practice.

(3) Such nation-wide standardization should not be based upon either compulsory national milk law or, except in the case of very small states, upon compulsory state milk law; but should instead be based upon voluntary local adoption by the cities and other local health districts of a uniform milk ordinance recommended by the national and state health officials. This policy is recommended for several reasons. (a) Our theory of government assumes that we promote the maximum practicable degree of local self government and reduce centralization of government to a minimum. Therefore, we should discourage centralization of milk control and, hence, milk control by state or federal law. (b) State enforcement of state regulations would, except in very small states, entail a larger state inspection force and, hence, a larger state appropriation than would be likely to be approved by the state legislature. (c) Joint enforcement of state regulations by both state and local milk inspectors would be certain to result in conflict and constant friction between the two groups of inspectors.

(4) The uniform requirements recommended by the federal and state health departments to the cities or local health districts should be such as to provide the local health officers with the most powerful enforcement devices. They should, therefore, provide for punishment of violations both by the degrading method and by the permit revocation method, as the health officer may elect. The recommended uniform local requirements should, therefore, be in the form of requirements for various grades of milk embracing the entire range of milk quality, rather than minimum requirements for all milk.

(5) In order to promote adequate local enforcement of the recommended uniform local regulations, the state should at intervals rate the milk control work of the local health departments. The ratings should be based upon a standard national rat-

ing method, such as the method devised and recommended by the Public Health Service.

(6) The state program for the public health supervision of milk supplies should be the function of the state health department.

(7) The uniform milk ordinance recommended by the federal and state health officials for local adoption and enforcement should be enforced in a uniform manner on the basis of the Public Health Service Milk Code.

(8) The milk sanitation records of the local health departments should be complete, and preferably kept upon standard forms similar to those which may be secured from the U. S. Government Printing Office.

(9) All health officials, federal, state, and local, should persistently recommend an adequate consumption of either Grade "A" pasteurized milk, or of certified or Grade "A" raw milk which has been pasteurized at home. The compulsory commercial pasteurization of all market milk is expedient and desirable only in communities in which the predominant sentiment is favorable. In other communities, an adequate period of consumer education should precede any action on this matter by the local governing body.

(10) In order that milk-borne outbreaks of disease may be most easily recognized, an adequate epidemiological method should be adopted by state and local health officials.

(11) The State Health Department of each state should carry out at least the following functions: (a) promote adoption of uniform local milk ordinances, (b) assist local health departments in training new milk control personnel, (c) assist local health departments in organizing the enforcement of the uniform ordinance, (d) provide expert advice on special technical problems, and (e) rate the milk sanitation work of each local health department at least once each year.

The above recommendations cover the essential points of the Public Health Service Milk Sanitation Program. They are given more fully with a complete discussion in "The Public Health Service Milk Sanitation Program," which was written by Sanitary Engineer Leslie C. Frank, and which is available for limited distribution in mimeographed form.

I. MILK-BORNE DISEASES

All pathogenic organisms found in milk come from some animal, or human, infected with the organism in question. Not every pathogenic organism which finds its way into milk, produces disease in the person who drinks it. The person may have a natural or acquired immunity for that organism. Or the organisms may be present in such small numbers that the person may be able to destroy them before a definite disease condition is set up. Not every contaminated milk supply produces an epidemic among the consumers. Even though disease may be produced, it may be limited to such few persons that no one suspects the milk supply.

Armstrong and Parran listed 791 American outbreaks of milk-borne diseases as reported in the literature from 1818 to 1927. However, the true number is much greater than this, since it includes only those outbreaks which found their way into the literature.

Since 1924 the Public Health Service has been sending annual questionnaires to all state health officers and to all city health officers of cities of over 10,000 population. These questionnaires have revealed 327 milk-borne outbreaks for the period 1924 to 1930 inclusive, or an average of approximately forty-seven per year. It it believed that there occur many more outbreaks which are actually milk-borne but which are not recognized as such.

The above discussion does not include the milk-borne diseases which do not usually occur in the form of epidemics, such as tuberculosis, undulant fever, and the intestinal disturbances of children. The public knows and hears less of these than of the much publicized epidemics.

Dr. Boswell, past president of the American Anti-Tuberculosis Society, has stated that 6 per cent of all fatal cases and 50 per cent of all non-fatal cases of tuberculosis are of bovine origin. He further stated that in 1930 there were 90,000 deaths in the United States from tuberculosis, or 5,400 from tuberculosis of bovine origin. There were 5,000,000 cases of tuberculosis in the United States in 1930, or 2,500,000 cases from bovine origin. Although the death rate from tuberculosis has decreased from 204 to 70, in the past 25 years, the above figures will indicate its seriousness both as a general public health problem and as a milk sanitation problem.

So far as I know, no one has been bold enough as yet to offer even an estimate of the deaths from intestinal disturbances which are caused by milk. But it is reasonable to assume that they have, at least in the past, been high. They almost invariably decrease as the quality and safety of a milk supply increases.

No one seems to know just how serious a public health problem undulant fever is, as yet. I shall not discuss it except to refer to a recently published paper by Murray, McNutt, and Purwin, of Iowa State College, on "The Effect of Pasteurization upon

Brucella." In their experiment, they used a 100-gallon coil pasteurizing vat. They show that a temperature of 143.6° to 145.4° F., applied 30 minutes, is sufficient to destroy Brucella organisms, both the bovine and porcine varieties. However, with the lid of the pasteurizer open, a much longer exposure to this temperature is required than when it is closed, and the results obtained were irregular and uncertain. Viable organisms were recovered from the foam and from a cold pocket, formed by an offset plug outlet valve, even after thirty minutes' pasteurization. I have merely mentioned this to show the importance of certain requirements which the Public Health Service has recommended for pasteurization.

II. ADVANTAGES OF NATION-WIDE STANDARDIZATION OF MILK SANITATION PRACTICE

A nation-wide standardization of milk sanitation practice will: (1) raise the average level of milk safety (this has been demonstrated by the Public Health Service Milk Ordinance); (2) simplify the milk sanitation enforcement work of the health officer; (3) increase milk consumption; (4) reduce the cost of milk sanitation to the producer and the consumer; and (5) by grouping several small communities, as all those in a county or district, it will enable communities that are too small to provide a milk control organization to secure the services of competent full-time inspectors.

III. ENFORCEMENT.

Assuming that the Public Health Service Milk Ordinance has been adopted by a city, what are the proper steps to take in enforcing it? The following suggestions are drawn from the experiences of the hundreds of cities now enforcing this ordinance:

(1) Immediately after the passage of the ordinance, the health officer or inspector should visit each dairy and pasteurization plant, study their present condition, and help them work out the best means by which they can meet the Grade "A" requirements of the ordinance. For this period, if the local inspector has not had previous experience in the enforcement of this ordinance, it will be advisable to ask the state department of health to detail a milk sanitarian for a short time to assist the local inspector until he becomes oriented in the work. I should like to insert a few "don'ts" at this point.

(a) Don't be too anxious to have every dairy graded "A" on the first announcement of grades.

(b) Don't award a dairy Grade "A" just because marked improvement has been made. Make the owner really earn the Grade "A" before you award it. If it is awarded before it is earned, you may never be able to secure correction of the defective items which have been overlooked. This will also tend to break down the respect of the dairyman for you and for the ordinance. It will probably force you to compromise with other dairymen and lead to endless requests for approval of changed plans, and to unnecessary arguments.

(c) Don't definitely say that no grades will be announced for six months or a year. Dairymen wait to see what the other fellow is going to do. They also wait until the last minute to start their own improvements.

(d) Don't talk to newspapermen or others about what you are going to do. After grades are announced, if you have done something worth talking about, don't miss an opportunity to tell about the safe milk supply which is available.

(e) Don't think that you can handle every dairyman alike and don't think that anyone can tell you how to handle each one.

(f) Don't just visit each dairy once, then wait until just before time for announcement of grades for the next visit, and expect the dairyman to have completed all the things you told him on the first visit.

(g) Don't depend entirely on bacterial counts to grade and degrade dairies.

Very few inspectors, new ones at least, use the milk control code sufficiently. Each inspector should know the code so that he does not need to consult it. In addition he should have a copy of the code with him on every inspection. The dairyman will know that the ordinance is based on a uniform plan and that, if he is awarded Grade "A," his dairy must conform with the same requirements that Grade "A" dairies in other and larger cities and in other states meet. He also should know that state and federal men may be expected to check up not only on his dairy but on the local inspector's work. Then if he wants to argue about any requirement, turn to the code and let him read what it says about that item. He still may not agree with what the code says, but you can use it to make him meet the requirements without making him feel hard toward you.

(2) When a sufficient period of time has elapsed so that a reasonable time has been allowed the dairymen to make the needed improvements, a definite date for the first announcement of grades should be set; the dairymen should be notified, and grades should be announced on that date.

(3) The dairymen who have failed to meet the requirements for Grade "A," for any reason, should be required to begin immediately to label their milk according to the grade to which they are entitled.

(4) If a dairy fails to meet the Grade "A" requirements on the first announcement of grades, but later corrects the defective items, the higher grade should be awarded as soon as it is earned.

(5) Continued supervision should be given the Grade "A" dairies so that items of sanitation may not be neglected either through ignorance, misunderstanding, or indifference on the part of the dairymen.

IV. PASTEURIZATION.

There can be no longer any question that authoritative and expert opinion is almost unanimously to the effect that all milk should be pasteurized. On the other hand, opinion is sharply divided as to whether pasteurization should be secured by compulsion or through education.

Where the majority of public opinion is for pasteurization, compulsion may be used; where the majority of public opinion is not for pasteurization or is definitely opposed to it, a program of education is probably the better. This educational program may be conducted through the newspapers, public meetings, and by personal contacts. Time does not permit the discussion which this question should have. I again refer you to the paper "The Public Health Service Milk Sanitation Program" for a more complete discussion.

V. THE PROMOTION OF MILK CONSUMPTION.

After the health officer has secured a proper compliance with the ordinance (but not before) he should take advantage of every opportunity to increase milk consumption to the optimum. As in the case of pasteurization, educational work on this item may take the form of radio or other addresses, newspaper articles, and pamphlets. Attention of the health officer to the cause of increased milk consumption should be as continuous and vigorous as attention to maintaining a safe milk supply.

COCOMALT IS ACCEPTED by the Committee on Foods of the American Medical Association. This food-drink is especially useful in the treatment of undernourished children and for expectant and nursing mothers. It is a scientific food concentrate produced by an exclusive process from barley malt extract, partially defatted chocolate and milk, sugar, whole eggs, flavoring and added Vitamin D. It comes in powder form, easy to mix with milk—hot or cold. So mixed, the result is a tempting, chocolate flavor food-drink—unusually high in caloric value yet easily digested and readily assimilated. Children love it. For samples, write R. B. Davis Co., Hoboken, N. J.

TRAUMATIC HEAD INJURIES

(Symposium at the Staff Meeting of the Good Samaritan Hospital, in Phoenix, Ariz., on June 27, 1932).

REVIEW OF RECENT LITERATURE

S. I. BLOOMHARDT, M. D.

MANAGEMENT OF ACUTE BRAIN INJURY

(Three Case Reports)

R. B. RANEY, M. D.

REVIEW OF HOSPITAL CASES

ROBT. S. FLINN, M. D.

(Dr. Bloomhardt)

Trotter divides the development of the treatment of cranial trauma cases into three periods; the first, extended from the earliest times up to approximately the middle of the nineteenth century. It was a time of slow progress unmarked by any striking discoveries, during which the sources of information were almost exclusively clinical and anatomical. The fracture of the skull was mostly stressed, the cerebral manifestations considered as complications of the fracture. Trephining over points of fracture was widely advocated, and very few of these cases were treated conservatively.

The next phase in the evolution of surgery of head injuries, dealt more with the fundamental problems of intracranial physiology and showed us the circulatory disturbance as the chief mechanism by which cerebral symptoms are produced, and it told us what symptoms are produced and how they are produced by a progressive lesion compressing the brain. In this period, the subject became so dominated by the theory of the localized compressing lesion that the student was apt to get the impression that the localized hemorrhage that lends itself to successful operative treatment was the usual form of head injury with which he would be called upon to deal. Such cases of local hemorrhage without any other serious injury do occur, but they are rare and form but a very small proportion of cases of head injuries observed in practice. This is the period in which many of us received our training. Let us give you Trotter's own words:

"The surgery of head injuries has inherited, from what we call its middle period, in addition to an invaluable treasure of the principles of cerebral physiology and pathology, a fallacious clinical impression that head injuries are largely a matter for urgent operation, which can and should be undertaken, in a considerable proportion of cases, with reasonable expectations of success. It is the melancholy duty of anyone making a seasonal survey of the subject to discourage this still rather widely prevalent view.

"The third, or present-day, concept of head injury places contusion of the brain as the commonest,

by far, of the serious lesions which follow cranial trauma. The amount of contusion may vary from a small localized cortical area to a wide-spread involvement producing patches of softening and hemorrhage throughout the brain. A cortical contusion cannot be repaired by surgery. A torn brain cannot be re-sutured. It is a realization of the facts that contusion is by far the most frequent result of severe cranial trauma and that surgery can accomplish nothing to repair the local damage, that has led to the present-day theory of the nonoperative treatment of head injuries unless the clinical evidence points unequivocally to the presence of a rapidly forming clot, or unless other methods of controlling intracranial pressure have failed".

Before one can treat head injury, he must have a conception of intracranial physiology. The brain is contained in a tight box of bones; namely, the skull. Enveloping the brain, within the skull, are the meninges. The cerebrospinal fluid surrounds the brain, circulating from the choroid plexus within the cerebral hemispheres outward over the cortex and channels formed between the surface of the brain and the arachnoid membrane. Following trauma, the brain swells, precisely as does any other part of the body. But as the brain swells, it at once encounters the confining cranial bones. The cerebral cortex is flattened against a vault, and the spaces through which the cerebrospinal fluid circulates are compressed. If the circulation is impeded, intracranial pressure is raised. If hemorrhage occurs, the contents of the skull are further increased and intracranial tension is elevated. Should the fracture be compound, especially if the meninges are torn, infection of the cerebrospinal fluid spaces can occur. The treatment of head injury is to prevent infection and to keep intracranial pressure within safe limits. If meningitis is avoided, and if the patient can be carried through the period of acute intracranial pressure from cerebral edema or hemorrhage, the outcome must be considered satisfactory. But, again let me state, laceration or contusion of the brain itself cannot be repaired by surgery. Head injuries are unquestionably increasing in frequency and the cause for this increase seems to be due to greater speed in transportation, as exemplified particularly by the automobile.

The review of the literature for the past year seems to show, without much question, that conservative methods in the treatment of cranial injuries are becoming generally adopted.

Vulliet, discussing results of this type of case in Switzerland, and Schuck, considering a group of head injuries encountered in Berlin—both comment on the lowering of the mortality rate following conservative treatment, in spite of the fact that the percentage of head cases to all hospital admission has steadily increased. Vulliet's mortality in a recent series treated conservatively was 20 per cent. Guynes and Hardy, reviewing cranial trauma cases in Texas, state that the mortality rate in 1926 was 75 per cent, in 1929 34.5 per cent. They believe that the lowering of the mortality resulted directly from the introduction of nonoperative treatment. Rand and Dougherty speak of lowered mortality rate in expectant treatment. Thorning reports a mortality of 23 per cent in his last 100 cases in a Houston clinic. Of these, there were twenty-three fatalities; fourteen died in less than thirty-six hours, eight in less than four hours. It is this type of cranial trauma, usually accompanied by other injuries, making them hopeless cases from the start, that provides an inevitable high mortality in the cerebral trauma group.

Weaver reviewed the result of treatment in 600 cases from cranial trauma. He did this in order to make a clinical study of head injuries in negroes as compared with head injuries in the white, to determine if there was any clinical foundation for the prevalent idea that a negro's head was more difficult to injure than a white man's. He took 300 cases of each, as nearly alike as possible; there were eighty-three deaths in the white and forty-eight in the black, making the death rate in the white 27.6 per cent, and in the negro 16 per cent.

"It is particularly noticeable that there were seventy-one massive injuries among the whites as compared with thirty-one injuries of the same type among the negroes, while there were forty-eight depressed fractures among the negroes and thirty-three among the whites. A lot of this may be accounted for by the fact that the force of the blow was expended in only fracturing the skull in the negro cases, while a massive injury resulted in the white cases—conclusive proof in itself that the negro's skull will withstand at least twice as much stress and strain as the skull of the white man." In the 600 cases, 102 had massive injuries, and all died but one; seventeen operations were performed in this group, with no recoveries. Here, again, is the group of patients who provide the inescapable mortality in head injuries. Nothing can be done for them. Weaver attempted only seventeen operations, apparently always as a last desperate resort. It is in dealing with this type of patient that the most pressure is put upon the surgeon to do something. Whenever he allows himself to be forced into hasty action, he regrets it afterward. There were but five cases of meningeal hemorrhage in the 600 head injuries, definite evidence of relative rarity of extradural clot formation as a result of cranial trauma. Four of these were operated upon and three recovered. Eighty-one cases of depressed fractures, with sixty-eight recoveries, form a third group. By far the largest group, 408 cases, were treated by expectant methods. None were operated upon and all but eleven recovered. It is possible that at least two-thirds of cranial trauma cases will recover without operation, under expectant treatment. Thorning states that he has yet to see a case of serious brain injury which was not accompanied by some degree of shock. It is not only absolutely futile, but positively wrong, to operate on these patients while they remain in a condition of shock.

they cannot rally without operation, they most certainly cannot with it. We must not depend upon pulse, respiration, or blood pressure findings, solely, nor upon temperature changes, solely; in fact, no one symptom or sign is of paramount importance. The whole picture must be considered; neurologic symptoms, state of consciousness, at times spinal fluid pressure, pulse, temperature, respiration, blood pressure—all must be taken into account. Some of the men are standing out strongly against lumbar puncture or hypertonic solutions by vein. Dandy states that the former may do harm and the latter does no good. Certainly a lot of harm can be done with the lumbar puncture, but it seems to be the consensus of opinion of some of our better head surgeons that, if it is properly performed, it is not dangerous and may be very beneficial. They state that by "properly performed" lumbar puncture, they mean a puncture performed by a manometric determination of the spinal pressure. They insist that a manometer should always be used, and, furthermore, the fluid should be removed in 1 cc. amounts, with the readings after each withdrawal. They claim it is folly to think that pressure may be estimated by counting the drops of fluid escaping from the needle.

Keegan claims contusion with more or less blood in the spinal fluid, is the common lesion and, since a bloody fluid is absorbed with difficulty from the subarachnoid spaces, he advises repeated lumbar puncture to aid in its removal, this more promptly restoring normal cerebrospinal fluid circulation. In this way, he believes that many of the post-traumatic headaches and low-grade pressure symptoms due to cerebral fluid stasis, may be avoided.

Thorning and Weaver emphasize the necessity for prompt debridement and closure of compound fractures, telling us that it is more important to treat compound fractures of the skull carefully and quickly than similar injuries of any of the long bones, for, if a patient has an infected compound fracture in an extremity, he may lose a limb, but if a compound skull fracture becomes infected, meningitis may result, with almost certain loss of life.

Investigation gives me the opinion that simple depressed fractures need not be promptly elevated. Whatever damage is done to the brain, occurs at the moment of impact. The presence of the depression itself does not increase the damage for some time, at least.

(Dr. Raney)

Acute brain injury is a broad term, but I prefer to use it rather than skull fracture, concussion, and so forth, because it covers a multitude of sins and includes everything from gross laceration of the brain substance to mild petechial hemorrhages that occur in simple uncomplicated concussions. The frequently used term of skull fracture does not completely cover the field that it represents, for it is possible to have a fractured skull uncomplicated, without injury to the brain, and such a condition is no more serious than fractures elsewhere in the body, and requires no treatment.

With our present-day means of rapid transportation, acute brain injuries have assumed a position of one of the most frequent injuries of the human body. Every physician practising, even in the rural districts, has had access to quite wide experiences in this field, in a lifetime of practice. The medical profession as a whole, in general practice or otherwise, demonstrate excellent judgment in the more common maladies and follow out their treatment with excellent precision. This being an actual fact, we wonder at times why such gross neglect exists so widely in the treatment of traumatic lesions of the brain. This is not only true of the medical profession, but it is even true of the standardized hospitals. I am not denying the fact that a hospital's standing depends upon the qualification of the members of its staff; however, it is a fact that an executive board exists, and through this medium daily gossip travels from corridor to corridor in the form of criticism, both constructive and destructive, which in turn influences to a certain extent the attention of patients. I have seen everything from petty squabbles to indignation meetings concerning an individual's judgment regarding curettage, cesarean section, and so forth. In fact, everything down to and including what might be considered the most effective dose of aspirin. This state of affairs has been going on for years, while, on the other hand, one of the largest contributing factors to every hospital mortality is being almost completely overlooked, excepting in those institutions with well-established neurosurgical sections. Such chronicity of undeviated neglectful procedure is commonly termed "being in a rut," and the only difference between a rut and a grave is that a rut is much longer. Consultation previous to cesarean section and curettage, in most hospitals today, has almost reached the point of being an axiom, while the acute brain injuries are operated promiscuously or are thrown into wards and allowed to die a natural death. If you don't believe this, examine your hospital records of acute brain injury and see for yourself. After all, hospital rules demanding consultation are for the purpose of saving lives, and if we are going to split hairs on conditions that have a very low mortality, why utterly neglect our most serious problems of the day?

Any brain injury, regardless of amount leaves behind a residue that is carried to the grave, ranging through gross insanity, epileptic seizures, headaches, personality changes, paralysis, changes in temperament, and so forth, depending upon the location and extent of the injury sustained. In treating acute injuries of the brain, if just one point be kept in mind, not only will the mortality be markedly diminished, but residual after-effects will be cut in half. Increased intracranial pressure following the shock period is the offending factor, and if not controlled, further destructive changes may go on even for years. Fortunately, the skulls are fractured in a number of the cases, and the patients are automatically decompressed, and if they are for-

tunate enough to escape meningeal infection and the original injury is not too extensive, they will get well if just left alone. Many a patient has failed to get by the crisis because shaken from x-ray to fracture room for the care of unimportant injuries, overlooking the major issue entirely.

Traumatic, increased intracranial pressure may occur from any of the following sources: intracranial hemorrhage, medullary edema (either the intra- or extra-medullary type), or all combined, plus depressed fracutres. The treatment of these conditions demands close attention and constant watching. Before attempting any type of surgical treatment whatsoever, shock, if present, should be controlled. After the control of shock, dehydration therapy should be established. This is usually done by fluid restriction and intravenous administration of concentrated solutions of glucose and magnesium sulphate. If this method fails, in association with frequent spinal punctures, surgery should be attempted before the patient enters the stage of medullary compression. After the stage of medullary compression has fully established itself, the mortality is 100 per cent. Decompressions during the first stages of compression may occasionally save a life; however, when definitely established, the outlook is almost hopeless. The following case history represents just such a case plus deceiving complications:

Case 1. Male, age 48, previous to injury good health, came under my observation three days after admission. Coma was profound, with intermittent attacks of convulsive mania. There was compound fracture of the femur and tibia on the right side, and a similar condition in the right arm. There was an extensive compound fracture of the right temporal, parietal and occipital bones, with depression over the squamous portion of the temporal bone (depression disclosed at surgical section). Temperature 104°, B.P. 150/70, pulse 60, respirations 18. Neurological Examination: Cranial nerve function could be only poorly interpreted, due to comatose condition. Reflexes: abdominal absent. Knee jerks: on left side absent. No pathological reflexes. Reflexes on the right side of the body could not be observed, due to trauma. Spinal fluid pressure, 48 mm. of mercury. Pressure reduced to 16 mm. of mercury. Fluid was bloody. Dehydration regime was established; all fluids discontinued. Glucose, 50 grams, in the vein. Two hours later, temperature dropped to 100°, pulse came up to 90, respirations became more regular, coma became much less profound, patient was talking incoherently; B.P. dropped considerably—120/60. Under vigorous dehydration and spinal punctures, increased intracranial pressure was fairly well controlled for 36 hours, at which time beginning medullary compression established itself within an hour. Temperature stepped up from 99° to 103°, pulse from 90 to 130, respirations from 25 to 40. Sub-temporal decompression: at this time a depressed fracture was found on the right side in the upper region of the squamous portion of the temporal bone; however, it had not lacerated the dura. The dura was opened; there was a very small amount of spinal fluid escaped, moderately blood-tinged, no gross hemorrhages. Convolutions of the brain were markedly edematous, with numerous petechial hemorrhages. Patient expired from acute medullary compression, 8 hours later. Autopsy: There was a masssive subdural hemorrhage from a rupture of the middle meningeal artery on the left side. No doubt, had there not existed extensive injury on the right side of the body, neurological findings would have disclosed the presence of this hemorrhage. The meningeal artery could have been ligated and possibly the patient's life saved.

Case 2. Male, previously in good health, immediately unconscious following a head injury for approximately 30 minutes, without shock when seen two hours later. There were no findings of importance. Complaint was a marked headache of a diffuse character. He had been struck on the left side of the head with a beer bottle. Spinal puncture, pressure 12 mm. of mercury; fluid bloody, pulse 80, B.P. 120/70, respirations 22, temperature 98.6°. Three hours later, pulse dropped to between 40 and 50, irregular. Respirations dropped to between 10 and 12, irregular, patient yawning. Left knee jerks sluggish; otherwise, neurological findings were absent. Dehydration therapy was established with fluid restriction and punctures. Twenty-six hours later there was no marked deviation from the original B.P. record. Pulse remained between 40 and 50 per minute and irregular. Respirations irregular and s ow. Temperature had slowly elevated to 102.8°. Yawning was quite evident. Knee jerks still sluggish, but a left positive Babinski had developed. No convulsions. Two hours later, patient had lapsed into unconsciousness. Right-side sub-temporal decompression: there was a massive sub-dural hemorrhage which was washed away gently with normal salt solution. When the pressure was relieved, coma cleared up while the patient was still on the operating table. Convalescence was uneventful. An interesting phenomenon in this patient is that he has had a choked disk for the last two years; however, he has no complaints otherwise, and has a profitable occupation. This is the type of a case in which the findings were on the opposite side of the head from the injury, not very marked, and could have been interpreted as a right-sided diffuse involvement. The classical signs of dilated pupil, pathological reflexes and convulsions do not always occur, and if they had been waited for in this case, death would probably have been the result, from an acute medullary compression. On the other hand, if medullary compression failed to establish itself and watchful waiting failed to produce the classical picture, irreparable damage would no doubt result.

The following case is quite illustrative:

Case 3 represents sequelae of long-standing pressure. Injury 9 years previous; 6 months later, patient developed left temporal headaches and convulsions, increasing in intensity. Neurological examination; double choked disk, left peripheral facial weakness, left lateral nystagmus; moderate impairment of 9th, 10th, 11th and 12th cranial nerves.

Patient had undergone considerable loss of memory and personality change. Neurological findings are confusing. Ventriculogram disclosed normal lateral ventricles, ruling out a posterior fossa-lesion, other than probably a displacement factor or residue of concussion. Left frontal osteoplastic flap, organized chronic massive sub-dural hematoma. Fifty cc. of fluid was removed and membrane was dissected away. No doubt this patient has been helped, for he has had no convulsions since operation; however, that is no sign that he has been relieved. The probability is that the convulsions will return, but will more than likely be milder and farther apart. Nine years ago a more favorable prognosis could have been given.

The three cases described bring out quite clearly some of the pitfalls encountered in early and essential diagnosis. If constant and close observation is applied, many of these pitfalls can be avoided and the ranks of the unknown soldier will not increase.

(Dr. Flinn)

This is a brief account of the cases of injury to the head treated in this hospital during the past 18 months. In all but two, there was definite fracture of the skull. However, "injury to the head' is a more inclusive term than simply skull fracture, for in a sense we are not much concerned with the fracture of the skull p-r se, but rather we are concerned with the amount of injury to the brain substance. In this regard, fractures of the skull differ in their importance from all other fractures of bony substance in the body. Inasmuch as patients do not die from fracture of the skull, but rather from injury to the brain, the treatment must be directed to the brain and not to the skull fracture itself.

From January 1, 1931, to June, 1932, twenty-one persons were admitted to this hospital suffering from injuries to the brain. A large majority of these injuries were inflicted by means of the motor car. Seventeen of the patients were males and four were females. In age distribution the patients varied from 3 years to 84, with an average age of 32.8.

Of the 21 patients admitted, 14, or 66.6 per cent, died. This mortality rate is considerably above that of 37.8 per cent of the 1000 cases recently reviewed by Foster Kennedy, and Dandy average of about 20 per cent.

Lumbar puncture was carried out in five cases. The fluid was bloody in four cases, all of which later died, and clear in one case, which recovered.

Operations undertaken numbered six. Four of these cases died, the mortality rate being 66.6 per cent, so that we may say that, the mortality rate for the operative cases being identical with that of the group as a whole, the patient's life was not jeopardized as a result of operative interference. Four elevations of depressed fractures were carried out, one trephine was done.

A somewhat casual perusal of the case reports shows that, on the whole, the cases of head injury have been treated in a careful and conservative fashion. Greater attention should be given to the neurological examinations so that a just appraisal of disability may be given. It would be well to summarize each case upon discharge, giving a careful account of the change in neurological findings from time to time. This is especially important in medico-legal cases. It is generally agreed that only a small minority of cases of head injury return to the hospital later with complaints. If, therefore, a patient is making a claim for disability on the ground of permanent injury, it is of great importance to know if there was history of loss of consciousness, convulsive states, focal cerebral palsies, and so forth.

When careful neurological notes are made, the question of definite disability or suspected post-traumatic neurosis can be definitely settled.

DALLAS SOUTHERN CLINICAL SOCIETY

The programs of the Annual Spring Clinical Conference of the Dallas Southern Clinical Society have been distributed through the mails. This Conference will be held at Dallas, March 27 to 31, in the week immediately following the Panamerican Medical Congress in the same city.

The guest speakers include the following: Dr. C. C. Sturgis, Professor of Medicine. University of Michigan; Dr. Lewellys F. Barker, Emerson Professor of Medicine at Johns Hopkins University; Dr. Joseph Brenneman, Professor of Pediatrics, Northwestern University; Dr. Percival Bailey, Professor of Surgery, University of Chicago; Dr. Chas. C. Dennie, Assistant Professor of Dermatology and Syphilology, University of Kansas; Dr. Joseph F. McCarthy, Professor of Urology, New York Postgraduate Medical School; Dr. A. E. Hertzler, Professor of Surgery, University of Kansas; Dr. Eugene R. Lewis, University of Southern California; Dr. Louis A. Buie, Sectioon on Proctology, Mayo Clinic; Dr. H. S. Crossen, Professor of Clinical Gynecology, Washington University; Dr. Philip H. Kreuscher, Professor of Traumatic Surgery, Loyola University (Chicago); Dr. Thos. B. Holloway, Professor of Ophthalmology, University of Pennsylvania.

The clinics, luncheons, round table discussions, public meetings, and postgraduate instruction periods, make this Conference a notable meeting. Reduced railroad rates on all roads. For further information, address, Secretary, Dallas Southern Clinical Society, Medical Arts Bldg., Dallas, Texas.

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ARIZONA STATE MEDICAL ASSOCIATION MEETING
(Tucson, April 20-22)

The forthcoming meeting of the Arizona State Medical Association to be held in Tucson, April 20 to 22, promises to be a notable one. In the first place, the Program Committee, with Dr. G. H. Fitzgerald, of Tucson, as Chairman, has the program practically complete two months in advance of the meeting. A tentative program is given below, and the complete program will appear in our March issue.

In the second place, grave issues face the medical practitioner and it is imperative that the thinking members of the profession shall meet and consider these with all the combined intelligence at their command. This is no time for any doctor to sulk in his tent and bewail his misfortunes.

In the third place, the best use for the spare time which many doctors find themselves blessed (or cursed) with is to improve our knowledge of medicine by seeking contact with other minds, and listening to the ideas of other people. It will be to our everlasting shame if we do not come through these years of trial better doctors. The best cure for the pain of a stock-market loss, or a reduced income, is the joy of learning something new, or a better way of doing our everyday tasks.

The tentative program contains the following papers, to which group others will be added:

Dr. James A. Dickson, of the Cleveland Clinic, Cleveland, O., will write on some orthopedic subject, the exact title not yet being announced.

Dr. Albert Soiland, of Los Angeles, will present the latest ideas on "The Diagnosis and Treatment of Cancer of the Breast."

There will be a symposium on Prostatitis, with the following papers:

Dr. Franklin Farman, of Los Angeles, will give a general presentation of the subject, using the title, "Prostatitis."

Dr. G. J. Thompson, of the Mayo Clinic, will write on "Prostatic Resection in Poor Risk Patients."

Dr. W. G. Shultz, of Tucson, will give a "Cinema Demonstration of Prostatic Resection."

Dr. Hal Rice, of Morenci, Ariz., will write on "Gas Gangrene."

Dr. E. A. Gatterdam, of Phoenix, will present the subject of "Oral Administration of Pollen Extract in Hay Fever Therapy."

Dr. W. Warner Watkins, of Phoenix, will present a paper on "X-ray Radiation of Acute Infections."

Dr. H. A. Reese, of Yuma, will have a paper on "Tularemia."

Dr. Emil C. Houle, of Nogales, Ariz., will write on "Belated Treatment of Sequelae of Diphtheria."

Dr. R. J. Stroud, of Tempe, Ariz., will discuss the subject of the Report of the Committee on the Costs of Medical Care under the title of "The New Experiment,—a Viewpoint."

Dr. L. B. Baldwin, of Phoenix, Ariz., will present the subject of "Some Clinical Aspects of Essential Hypertension."

Dr. John W. Flinn, of Prsecott, Ariz., will discuss the subject of "Bone and Joint Tuberculosis."

Dr. Victor Randolph, of Phoenix, will discuss the subject of "Intrapleural Pneumolysis, with Special Reference to Bilateral Pneumothorax, in the Treatment of Tuberculosis."

Dr. Walter P. Sherrill, of Phoenix, will have a paper on the subject of "Tuberculosis from the Standpoint of the Pediatrician."

Dr. Henry L. Franklin, of Phoenix, will have a

paper on "Refractive Errors in Their Relation to General Medicine."

Dr. Robt. Allen Hicks, of Tucson, will discuss a "Progress Chart for the Study of Pulmonary Tuberculosis as Elaborated at the Southern Pacific Sanatorium."

Dr. Howell Randolph, of Phoenix, will show a movie illustrating the "Mechanism of Electrocardiography and the Heart Beat."

Dr. Clara S. Webster, of Tucson, will have a paper, title of which is not yet announced.

The Pima County Medical Society expect to make this meeting a memorable one, and will curtail no effort looking toward this objective. The committees of the local society who will govern the various activities of the meeting are the following:

Advisory Committee: N. C. Bledsoe, S. H. Watson, Dan L. Mahoney, J. B. Littlefield and Alvin Kirmse.

Finance Committee: S. H. Watson.

Smoker Committee: J. B. Littlefield and Alvin Kirmse.

Dinner-Dance Committee: G. S. Kibler, W. Paul Holbrook and E. J. Gotthelf.

Reception Committee: Dan L. Mahoney, S. H. James, C. A. Thomas, Allen K. Krause, V. M. Gore and S. H. Watson.

Clinics Committee: V. A. Smelker, C. A. Thomas, Meade Clyne, R. A. Davison, M. C. Comer and J. J. Beatty.

Exhibits Committee: J. J. Beatty, J. L. McKnight, W. G. Shultz, E. M. Hayden and R. A. Hicks.

Ladies' Entertainment: Pima County Society Auxiliary.

The officers of the State Association are:

President—Clarence Gunter, Globe.

President-Elect—N. C. Bledsoe, Tucson.

Vice-President—C. W. Swackhamer, Superior.

Secretary—D. F. Harbridge, Phoenix.

Treasurer—C. E. Yount, Prescott.

Councilors—C. A. Thomas (Tucson), Victor Randolph (Phoenix), F. P. Manning, (Flagstaff).

The Committee on Scientific Program consists of G. F. Fitzgerald of Tucson, with N. C. Bledsoe and D. F. Harbridge, ex-officio members.

The hotel headquarters will be the Santa Rita Hotel. The general scientific meetings will be in the Scottish Rite temple auditorium.

EL PASO COUNTY MEDICAL SOCIETY

The newly elected officers of the Society for the year 1933 are as follows:

President: James A. Pickett.

Vice President: James J. Gorman.

Secretary-Treasurer: Leslie Smith.

Associate Editor: W. E. Vandevere.

MARICOPA COUNTY MEDICAL SOCIETY (Ariz.)

The officers of this Society, elected at the December meeting were as follows:

President: A. M. Tuthill.

Vice President: F. C. Jordan.

Secretary-Treasurer: O. W. Thoeny.

Censor: S. I. Bloomhardt.

Directors: J. J. McLoone and H. D. Ketcherside (Note: Dr. McLoone later resigned and Dr. Geo. Truman of Mesa was selected to fill the vacancy.)

PUBLIC HEALTH NOTES

J. ROSSLYN EARP, Dr. P. H.
Director New Mexico State Bureau of Public Health.

PYRETHRUM

Much has been written regarding the war between insects and man. There are those who hold that the issue is even now undetermined. Can human intelligence avail against the socialized activities, the enormous breeding power, the economic efficiency of the insect world?

Suppose that a drug should be discovered which was at once deadly to insects and harmless to man? Such a drug has, in fact, been in use for centuries in the orient, and we scientific people of the west are just beginning to awaken to its possibilities. Now that we are getting to work, we are going to use it far more effectively than it has ever been used before.

The drug is pyrethron, an ester formed by combination of the base pyrethrolone with either chrysanthemum monocarbonic acid or with chrysanthemum dicarbonic acid. The formulae will be found in Solis-Cohen and Githens' Pharmacotherapeutics.[1]

Of course the people of the orient did not know all this! To them the poison was just powdered Pyrethrum flowers—the same powder that is sold here as "insect powder." It is to two Swiss pharmacists, Von H. Standinger and Ruzicka,[2] that we owe the isolation

of the toxic principle, a poison which has been shown by Perrot[3] and his French collaborators to affect not only insects but the whole cold-blooded kingdom of animals, from frogs to the protozoa; not only insects, but also worms. And so we may conquer another important army of enemies of the public health. The action of pyrethron on frogs resembles in some respects that of strychnine or, even more closely, that of veratrine. But, whereas, strychnine and veratrine are also poisonous to warm-blooded animals, pyrethron can be given in large doses to dogs and rabbits[4] without causing them any inconvenience whatever.

The effects of pyrethron on insects is lethal, but more rapidly on some insects than on others. A table showing the effects of a solution of pyrethron known as "flytox"—a product well known in this country —on ten different insects is published in a paper by Abbatucci and Roubaud.[5] From this it appears that fleas and mosquitoes are the most sensitive of the insects tested, and cock-roaches the most resistant. One suspects that the ratio of body surface to weight may have something to do with this relative susceptibility.

Pyrethron is soluble in alcohol, sulphuric ether, ethylene, all of which solvents have disadvantages. The most satisfactory solvent for insecticide purposes so far discovered is plain kerosene. The writer made up a solution last fall of one pound of powdered pyrethrum in one gallon of kerosene and experimented with it upon the hardy flies of Canyon Road, Santa Fe. He found it to be at least as efficacious as any commercial preparation costing more than twice as much. It is quite probable that a smaller amount of pyrethrum would suffice, but exact dosage cannot readily be determined. "Dalmatian insect powder" is more potent than "Persian insect powder." It must be remembered also that the chrysanthemum carbonic acids are very weak and pyrethron is consequently an unstable compound. If pyrethrum powder is exposed to moist air, it rapidly loses its virtue. Solutions containing basic substances break up the compound very rapidly and destroy its activity. For this reason, attempts to use pyrethrum in a saponified emulsion have met with many failures. Kerosene solutions will undoubtedly be used in agriculture and are likely to displace the dangerous lead arsenate sprays, and so prove once again of value to public health.

As for the use of pyrethron as a vermifuge, it is full of possibility. One pyrethron vermifuge at least, by the name of Vermosal, is already on sale by the Societe Anonyme des Vermenes, 46 rue du Bac Asnieres, Seine, France. With this we have had no opportunity to experiment.

One word more (if the editor has space). The plants from which pyrethrum powder is obtained, namely Pyrethrum roseum, Pyrethrum carneum and Chrysanthemum cinerariaefolium, are not difficult to cultivate. The French trade is already almost, if not quite, able to supply itself with French-grown raw materials. There must be many places in America where these plants could be profitably grown instead of other agricultural products which are in excess of the demand. And it is my guess that the demand for pyrethrum will be a large and an increasing one.

REFERENCES

1. Solis-Cohen (S.) & Githens (T. S.): Pharmacotherapeutics, New York, 1928, pp. 437, 438.
2. Staudinger and Ruzick: Uber Isolierung and Konstitution des wirksamen Teiles des Dalmatinischen Insektenpulvers. Helvetica Chemica Acta 7: 177 and 377, 1924.
3. Perrot, E.: Le pyrethre et ses applications, Bull. de Sci. Pharm. 39: 42 (Jan) 1932.
4. Henry, A.; and Ripert, J,: Le pyrethre insecticide, Rec. de med. Vet., 103: 290, 1927.
5. Abbatucci, S.; and Roubaud, E.: Experiences sur un liquide insecticide commercial a base de petrole et de poudre d pyrethre, Bull. Soc. path. exot. 19: 901, 1926.

DISTRICT CONFERENCE OF THE AMERICAN COLLEGE OF SURGEONS

Hotel Westward Ho, Phoenix, Ariz.
March 27 and 28, 1933

OUTLINE OF SESSIONS

Monday, March 27

8:00— 9:00 Registration, Lobby, Hotel Westward Ho.
9:00—12:00 Clinics, Good Samaritan Hospital and St. Joseph's Hospital.
9:00—12:00 Hospital Conference, Main Dining Room of Hotel Westward Ho.
12:30— 2:00 Medical Motion Pictures, Main Dining Room of Hotel Westward Ho.
2:30— 5:00 Hospital Conference, Main Dining Room of Hotel.
5:00— 5:30 Annual Meeting Fellows of College, Main Dining Room.
8:00—10:30 Scientific Session, Eye, Ear, Nose and Throat, in Conference Room A, Mezzanine Floor, Hotel Westward Ho.
8:00—10:30 Scientific Session, General Surgery, Main Dining Room.

Tuesday, March 28

9:00—12:00 Clinics, Good Samaritan Hospital and St. Joseph's Hospital.
9:00—12:00 Hospital Conference, Good Samaritan Hospital Assembly Room.
12:30— 2:00 Medical Motion Pictures, Main Dining Room of Hotel.

2:00— 5:00 Hospital Conference, St. Joseph's Hospital Nurses' Class Room.
2:30— 5:00 Scientific Session, General Surgery, Main Dining Room of Hotel.
2:30— 5:00 Scientific Session, Eye, Ear, Nose and Throat, in Conference Room A, Hotel.
8:00—10:00 Community Health Meeting, Phoenix Union High School Auditorium.

VISITING SPEAKERS

Dr. Franklin H. Martin, Chicago. Director General, American College of Surgeons.

Dr. J. Bentley Squier, New York. Professor of Urology, Columbia University College of Physicians and Surgeons; President, American College of Surgeons.

Dr. William D. Haggard, Nashville. Professor of Clinical Surgery, Vanderbilt University School of Medicine; President-Elect, American College of Surgeons.

Dr. Charles H. Mayo, Rochester, Minnesota. Professor of Surgery, University of Minnesota Medical School and Graduate School, Mayo Foundation; Regent, American College of Surgeons.

Dr. Clarence Cook Little, New York. Managing Director, American Society for the Control of Cancer.

Dr. William V. Mullin, Cleveland. Oto-Laryngologist, Cleveland Clinic.

Dr. Alfred W. Adson, Rochester, Minnesota. Professor of Neurosurgery, Mayo Foundation.

Dr. Robert Jolly, Houston. Superintendent, Memorial Hospital.

Dr. Bowman C. Crowell, Chicago. Associate Director, American College of Surgeons, and Director of Clinical Research.

Dr. Malcolm T. MacEachern, Chicago. Associate Director, American College of Surgeons, and Director of Hospital Activities.

Dr. Geo. Crile, Cleveland.

More detailed program will be mailed out generally to the physicians and surgeons of the Southwest early in March.

CORRESPONDENCE

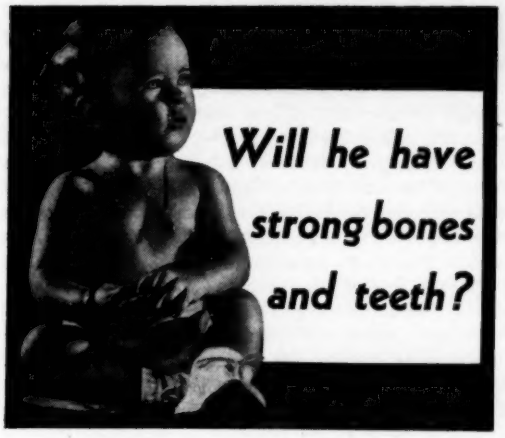
Winslow, Arizona,
January 25, 1933.

Southwestern Medicine,
Phoenix, Arizona.
Gentlemen:

The Navajo-Apache County Medical Association wishes to impart to all interested parties and for the good of the medical profession in general the activities of one R. N. Trotskey, who attempted to practice medicine in Winslow, Arizona, without first securing a license to do so.

Mr. Trotskey arrived in Winslow some time in the month of July, 1931, and represented that he was a graduate of several European medical schools; that he had a license to practice in the State of Arizona, and that he professed to specialize in ear, eye, nose and throat surgery.

It was soon discovered that Thotskey had no



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license to practice in the State of Arizona and apparently had no credentials or diploma from any medical school. The County Attorney of Navajo County filed a criminal complaint against him charging the crime of practicing medicine without a license and being tried before jury was convicted of the charge. Another complaint charging the same crime was filed against him and while out on bond pending the hearing on the second charge he removed one of his patients from Winslow, Arizona to Flagstaff, Arizona and attempted to remove a fibroid tumor; the patient died and the post-mortem examination showed a lack of skill to perform such operations. He was tried before a jury in Coconino County for the crime of murder but was acquitted although the prosecution showed that Trotskey performed the operation, that he was not admitted to practice surgery or medicine in the State of Arizona; and that the patient died as the result of the operation.

We understand that now Trotskey is lecturing from Station K. S. L. at Salt Lake City, Utah and is posing as an ear, eye, nose and throat specialist.

His record under the following aliases is on record in the U. S. Department of Justice, Bureau of Investigation, Washington, D. C., should you wish to inquire: Ray Anthony, Rob Luken, Romano Trosky, Romano N. Trotzky, Romano Trotsky.

It is sincerely hoped that you will take the necessary action to inform the various Boards of Medical Examiners of the activities of this man.

Yours truly,

J. W. BAZELL, M. D.
Secretary of Navajo-Apache
County Medical Association.

A RESOLUTION

TO THE SECRETARIES OF THE COUNTY MEDICAL SOCIETIES:

The following resolution was passed by the Fulton County and Cobb County (Georgia) Medical Societies at their regular meetings a few weeks ago, after the reading of a paper entitled "The Economic Status of the Medical Profession," which appeared later in the Journal of the American Medical Association, October 15, 1932, Page 1358, under the Department of Medical Economics.

Since this article appeared, we have had numerous letters from physicians all over the United States urging that this plan be placed before the different county, state and national societies for their consideration and adoption.

We are asking that you place this or a similar resolution before your society and ask for its adoption. Have it signed by your proper officials and forward to the secretary of the Medical Association of your state.

These resolutions will be placed in the hands of the delegates to the American Medical Association at its next annual meeting.

RESOLUTION

Whereas, the medical profession of the United States is suffering losses all out of proportion to its ability to endure, and

Whereas, our losses are not so much due to the financial depression as to over production of doctors and decreased demand for paid medical services generally, and

Whereas, the production of doctors is far in excess of the population increase,

Therefore, we the members of the..... County Medical Society respectfully request the Medical Association of ....., the other State Medical Associations, the Southern Medical Association and the American Medical Association to adopt this or similar resolutions requesting the Medical Colleges of the United States to reduce the number of graduates each year until the law of supply and demand has been fully complied with. The number of graduates to be determined by a national committee appointed by the President of the American Medical Association.

Respectfully submitted,

.....M. D., President
.....M. D., Secretary.

ACHIEVEMENTS IN MEDICINE DRAMATIZED IN NEW RADIO SERIES

On January 8, the House of E. R. Squibb and Sons presented the first of a series of half-hour radio programs in keeping with the splendid traditions of its founder.

This half hour of entertainment is on the air every Sunday over the Red Net Work of the National Broadcasting Company Chain, at 4:30 p. m. New York time. It features Frank Black and his Orchestra, the Revelers, and as the high spot a dramatization of gripping moments from the history of medicine.

These presentations of music and interesting dramatic episodes are designed to appeal to almost

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every type of radio listener. The announcements emphasize that only through a sufficient number of properly trained physicians can a community expect to meet its responsibility for the care and prevention of illness and the protection of health. Impressive reasons are also mentioned as to why the use of the family doctor is a good way to keep down the costs of competent, sympathetic, and understanding general medical care.

ORAL THERAPY IN CONGENITAL SYPHILIS

The use of Stovarsol in congenital syphilis by pediatricians abroad induced Maxwell and Glaser to investigate the value of the therapy. The results achieved in ten cases are reported in full in the Am. J. Dis. Child. 43:1461-1489, June, 1932.

In four cases the Wassermann reactions were negative when treatment was begun but as the parents were 4+ treatment was not delayed. These cases had remained negative over a period of thirteen months when reported. The other six cases had positive Wassermann reactions. Three were reversed after the first course of treatment, two at the end of the first rest period, and the other at the end of the second course of Stovarsol therapy. The results in these infants, all under one year of age when treatment was begun, are encouraging. Children over one year of age did not respond so readily.

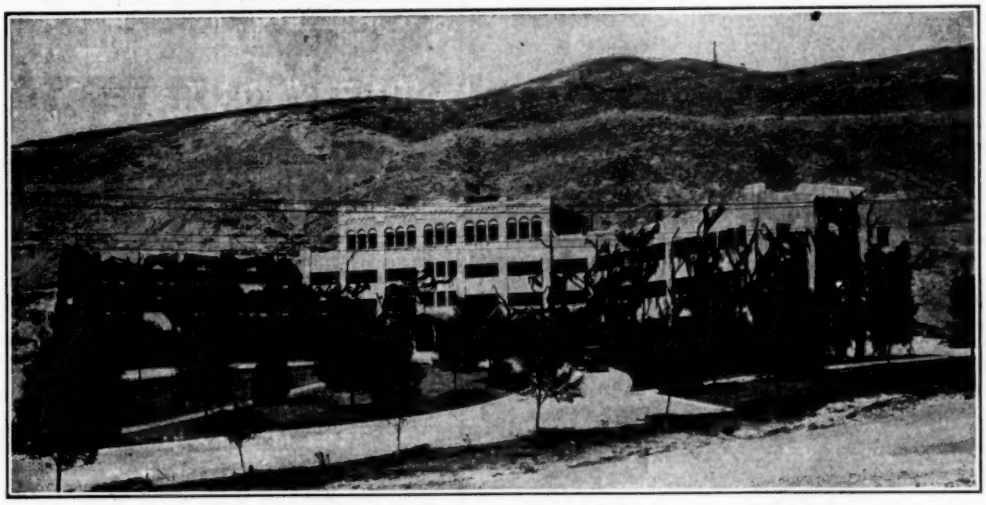
Other advantageous features of Stovarsol therapy in congenital syphilis in infants and children as reported are: the remedy is administered by mouth, and the patients generally show improvement in appetite, general vigor and energy.

The authors believe that Stovarsol has a definite place in the treatment for congenital syphilis. Administration must always be under the direction of the physician as toxic symptoms may appear. In the majority of cases these symptoms are evidently mild in character but occasionally they may be severe.

Stovarsol is a pentavalent arsenic preparation allied to arsphenamine in its chemical constitution. It is manufactured in this country by Messrs. Merck & Co., Inc., of Rahway, N. J.

COMMERCIALISM VS. PROFESSIONALISM

Under this title the Journal of the Medical Society of New Jersey relates the story of "One of the large manufacturers of a product extensively prescribed by practicing physicians, and extensively used for that reason, was, it seems, told by the representative of a powerful drug chain organization that it must make certain concessions to 'modern merchandising methods', or else . . . The first thing wanted was an extra discount. This was needed in order that the products in question might be advertised to the public. The advertising was deemed necessary in the interest of both the manufacturer and the chain organization which proposed to handle the products in question. The answer of the manufacturer was that he did not



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desire to advertise his products to the public. He prided himself that he advertised only to the medical profession. He felt that the public had no business prescribing for itself, even such a good product as he made.

"The retailer urged that goods such as those in question could be sold in larger quantities by displaying them in windows, advertising them in newspapers, and by the recommendation of clerks. The manufacturer did not want any of those procedures followed."

Such occurrences are an old story to ethical manufacturers, although the practicing physician doubtless is not aware of how much pressure is thus brought to bear.

S. M. A. Corporation ont only refuses to participate in such unethical practices, but in addition every package of S. M. A. from the beginning has borne this injunction: "Use only on order and under supervision of a licensed physician. He will give you instructions."

BOOK REVIEW

CLINICAL ENDOCRINOLOGY OF THE FEMALE, by Charles Mazer, M. D., F.A.C.S.; Assistant Professor of Gynecology and Obstetrics, Graduate School of Medicine, University of Pennsylvania; Gynecologist to Mt. Sinai and to Northern Liberties Hospitals, Philadelphia; and Leopold Goldstein, M. D.; Demonstrator of Obstetrics, Jefferson Medical College; Assistant Gynecologist to Mt. Sinai Hospital; Formerly Fellow in Gynecologic Research, University of Pennsylvania, Philadelphia. Illustrated. W. B. Saunders Company, Philadelphia and London; 1932.

Because of the hormone test of pregnancy and some of the other new ideas of endocrines in connection with the female, this book is particularly interesting. The book is built from the literature. It has 649 references. It is written in a clear, understandable style. It is profusely illustrated. The text seems thoroughly reliable. The reviewer has no hesitancy in recommending it as a scientific treatise on the subject. O. H. B.

DIAGNOSIS AND TREATMENT OF DISEASES OF THE THYROID GLAND: by George Crile and Associates. Philadelphia and London: W. B. Saunders Company, 1932, Cloth, $6.50 net.

This is a medium size book of 508 pages with an apparently excellent index and 164 illustrations. There are 39 chapters and 24 contributors.

Chapter Three deals with "The Role of the Thyroid Gland in the Energy System.' It is only four pages in length but it is a masterpiece from the pen of George Crile.

Particular stress is laid throughout the book upon diagnosis and differential diagnosis.

At the end of most of the chapters there is a paragraph or several short paragraphs with a summary of the chapter. This is a commendable practice which more authors should follow.

The book has many case reports illustrating various conditions of the thyroid or associated conditions. The illustrations are frequently photographs of patients before and after treatment.

Chapter 34 deals with "The Adrenal Factor in Hyperthyroidism and Its Control." This will interest many of the internists and surgeons dealing with the thyroid and other endocrine abnormalties.

An innovation in the book are chapters by nurses. Chapter 27 is by Abbie Porter on "The Role of the Nurse in the Preoperative and Postoperative Care of the Patient with Hyperthyroidism." Chapter 28 is by Emma M. Barr, on "The Role of the Operating Room Nurse in Operations on the Thyroid Gland." Chapter 29 is by Lou E. Adams on "The Advantages of Nitrous Oxide-oxygen Analgesia for Operations on the Thyroid Gland."

The work is unusually complete and presents the procedure followed at the Crile Clinic.

There are relatively few typographical errors. On page 87 in the third paragraph speaking of cases in which thyroidectomies had been performed the author uses thyroidectomy in the singular whereas his subject is plural.

The printers art is beautifully exemplified. The book is deserving of extensive distribution.

O. H. B.

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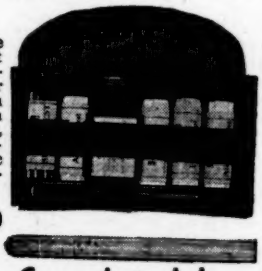

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